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Estd. 1904

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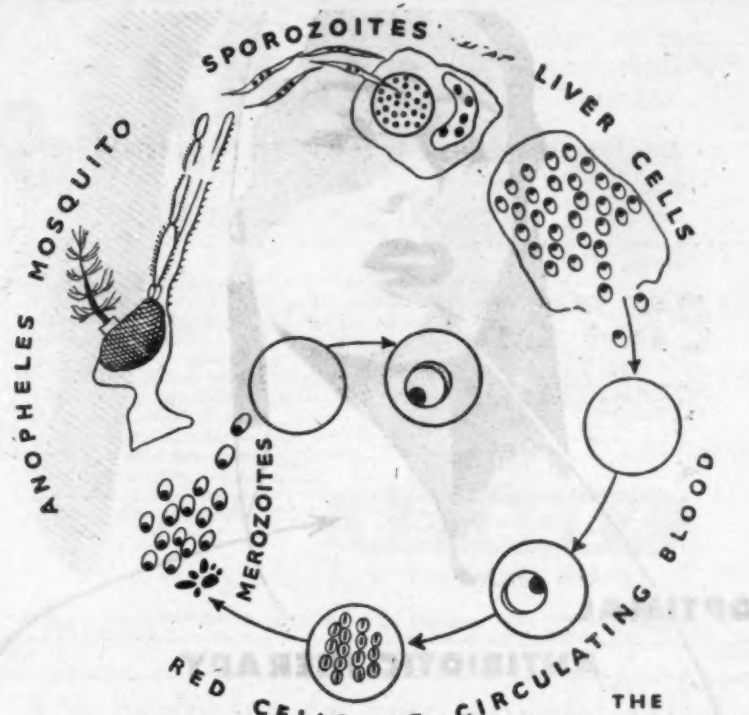

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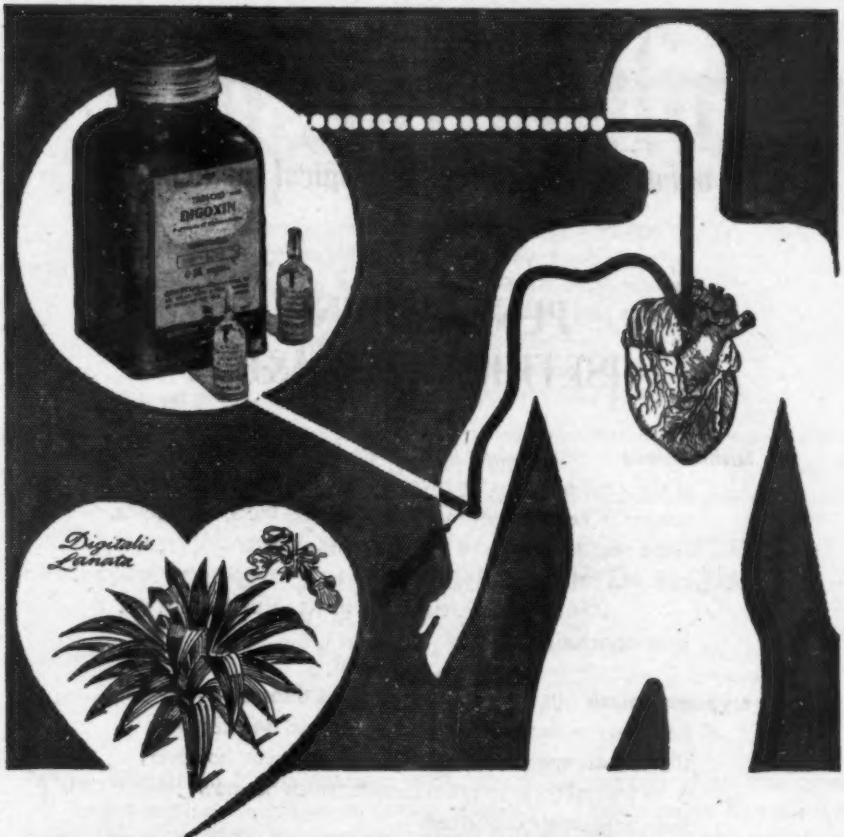

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STUDIES ON THE EFFECTS OF PROTEIN DEFICIENCY AMONG CHILDREN IN MADRAS

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AND

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Research Fellow.

THE transition from breast milk to adult mixed diet has been beset with difficulties in every country. Cow's milk has always been the chief item during the period but in less fortunate countries like India at present or at least parts of this country where there has been acute scarcity of milk during recent years, the problem has been baffling with dire consequences.

Infants and children with œdema all over the body without albuminuria have been admitted in large numbers in the past few years in the Childrens' Wards of the General Hospital and of the Women and Children Hospital, Madras, under the care of the senior author. In recent times they form about 9% of the total admissions (224 out of 2490 admissions in 1950). They come in œdematous, lack liveliness and seem to take little interest in their surroundings. A good proportion of them have skin changes (dermatoses of various types—denuded superficial layers, purpuric areas, scaly skin of pellagra). Many have keratomalacia, orogenital syndrome, anæmia and sparse light hair (*Figures 1, 2 and 3*). Diarrhœa is often present and food tolerance especially to fats is broken down. Many of these cases present a pitiable sight. The mortality was rather high in the earlier series but is much less now (16%) with blood and serum

transfusions to supplement the oral half cream or acid milk feeding, fortified by casein products and other proteids. Relapses are very common because the child gets the same old deficient diet at home after discharge from the hospital. A series of 78 cases was reviewed by one of us (Achar) in recent times. A further series of 150 cases investigated during the past 2 years by us revealed the following clinical features :—

Analysis of clinical features of 150 nutritional dystrophy cases

Oedema present in all—gross œdema in 50—moderate œdema in 60—very little in 3 (dry type).

Dermatoses (crazy-pavement or mosaic skin, denuded superficial layers, cracks and fissures at clexures, purpuric areas, depigmented or hyperpigmented areas, scaly skin of pellagra)	...	76 (51%)
Oral changes (cheilosis, angular stomatitis, glossitis)	...	110 (73%)
Eye changes (xerosis, keratomalacia, panophthalmitis, corneal opacity)	...	56 (37%)
Changes in hair (light hair, dry and brittle sparse hair)	...	87 (58%)
Palpable liver	...	97 (65%)
Intestinal infestation with ascaris lumbricoides	...	74 (49%)
Diarrhœa in hospital	...	140 (93%)
Mucus in stools during stay in hospital	...	42 (28%)
Amœba found in stools	...	5 (3%)
Previous history of dysentery	...	50 (33%)
Previous history of diarrhœa	...	29 (20%)

Age incidence:—Under 1 year :–3 (2%). Between 1 and 3 :–80 (53%). Between 3 and 5 :–56 (37%). Above 5 :–11 (7%).

In the previous review by one of us (1950) the pathological changes found in the liver at autopsy in 12 cases were described. There was extreme fatty infiltration with commencing periportal fibrosis in 2, while in 1 case there was diffuse fibrosis round the lobules. In the present series, biopsy studies were made in seventy cases by needle puncture of the liver, using a Silverman needle. In 18 of these cases, the histological changes in the liver subsequent to treatment were studied by repeat biopsy 2 to 3 weeks later. In addition we could study the liver at autopsy in 8 cases and the pancreas at autopsy in 3 cases. This study, the details of which are given in a separate paper under publication, revealed that there were constant pathological findings. Extensive fatty change was found in most of the autopsy and biopsy specimens, the tissue sometimes resembling adipose tissue rather than liver tissue (*vide Micro-photograph 1*). The liver was enlarged and palpable in 65% of the cases but even in those cases where the liver was not palpable biopsy studies revealed histo-pathological changes in the liver. There was a varying degree of fibrosis in a great majority of the liver specimens studied. Three of the cases showed diffuse

Studies on the Effects of Protein Deficiency among Children in Madras.

S. T. Ashir and V. Benjamin.

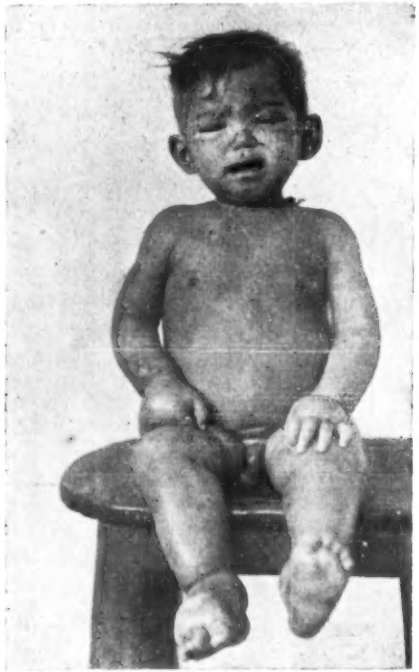

Fig. 1. Nutritional oedema of a marked degree.

Fig. 2. Note dermatoses (purpuric areas and denuded skin)

Fig. 3. Note Oedema and hair.

Studies on the Effects of Protein Deficiency among Children in Madras.

S. T. Achar and V. Benjamin.

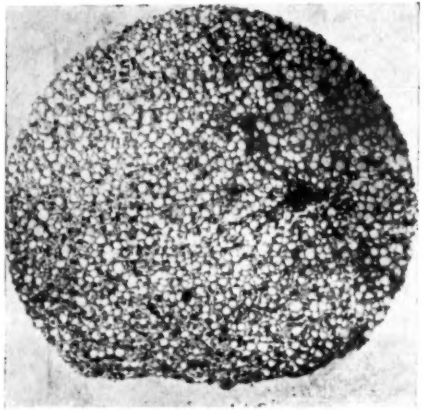

Fig. 1. Section of liver (biopsy specimen). Note well marked fat vacuolation of liver cells.

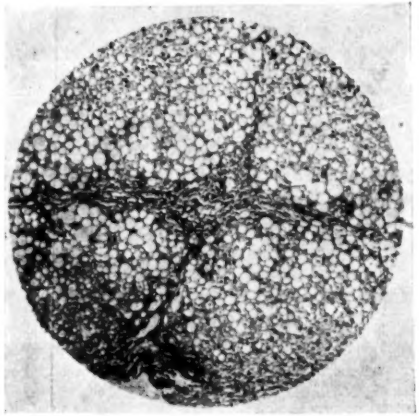

Fig. 2. Section of liver (biopsy). Early periportal fibroblastic reaction.

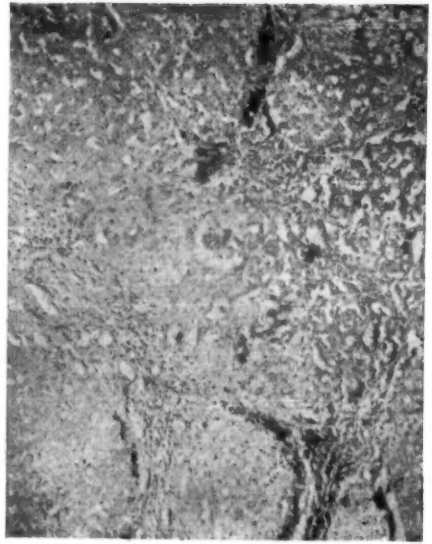

Fig. 3. Section of liver (autopsy). Marked periportal fibrosis.

hepatic fibrosis, one of them was only a two-year old child. (*vide Micro-photograph 3*). The pancreas showed well marked fibrosis and acinar atrophy in the 3 cases in which this was studied at autopsy.

The serum protein was estimated in 40 cases by the micro-keldajhl method and showed in all a diminution of the total protein, the average being 4·38 grams %. The serum albumin was diminished being below 3 grams in over 90 % of the cases and below 2 grams % in 40% of the cases. The albumin/globulin ratio was altered in over 75% of the cases. Under treatment by high oral proteid, supplemented by blood and serum transfusions, there was a rapid restoration to the normal pattern with clinical improvement in the 15 cases in which repeat serum protein estimations were carried out, but the liver histology showed little improvement on repeat biopsy in these cases. These studies show that while the serum protein improves quickly under the treatment the liver histology seems much slower in showing improvement and bears no direct relation to the levels of total protein and albumin on admission or to the degree of subsequent rise under treatment. Also the liver histology gave no clue to prognosis in the six cases which died, as compared to the histology of those that improved among the 70 cases in which liver biopsy studies were made.

The immediate effects of this condition seem to be:

1. Death in some cases, the mode of death suggesting an acute hepatic parenchymal failure.
2. Susceptibility to infection (ear, lung and skin infections).
3. Loss of vision due to keratomalacia.

Long term effects seem to be: (*a*) retarded growth and physical development, (*b*) a possible culmination in portal cirrhosis of some of the survivors the evidence for this is provided by some of the biopsy pictures of the liver (*vide Micro-photograph 3*) and by the relatively high incidence of portal cirrhosis of liver in Madras among the juveniles and adolescents (16% of all the cirrhosis liver admissions in the General Hospital in the past 5 years are below 20 years of age).

A survey of the incidence and distribution of this condition in Madras reveals that it is mostly amongst the poorer classes; the dietetic history of these children reveals a gross inadequacy of protein in their diets and in the great majority of the cases, cow's milk in any form was not given to these children. It is beyond doubt that the cause of this condition is lack of protein in the diet of the children, both quantitative and qualitative, and a rough survey of the diets consumed by these children revealed that the protein intake averaged ½—¾ gram per pound of expected body weight as against the 1½ grams or more of proteid per pound

body weight recommended by most nutritional experts for children of these ages. Qualitatively also the proteins consumed by these children were mainly from vegetable sources, from cereals and rice, meat being a costly and rare item. This condition of nutritional dystrophy has hardly been met with among breast-fed infants during the period of adequate breast-feeding. Mothers are sometimes able to adequately breast-feed upto $1^1/_2$ years, and little else is given as supplement. Such children, though exhibiting anæmia, do not develop the full-blown Kwashiorkor (the African name for the disease). It is also significant that no cases have been admitted from among the fishermen's children, and, on enquiry and survey made in the various poorer areas including the fishermen's areas, it was found that these fishermen children were started after weaning, on fish along with rice, usually from the age of one year. There were a few cases from middle classes and the richer classes also, and it was found in all these cases that there was a deficiency of protein intake from ignorance often combined with breakdown of milk tolerance after dysentery or diarrhœa, a feature commonly met with. A period of diarrhœa or dysentery often precipitated the onset of œdema. Presumably the breakdown of food tolerance and intestinal hypermotility interfered with digestion and absorption of food, precipitating the manifestations of protein deficiency. The common practice of giving children with diarrhœa only barley water and arrow-root conjee with very little protein over long periods was responsible in some of these cases for this condition.

In view of the constant liver changes as studied by biopsy in this series, a survey was made of apparently healthy children of some of the poorer areas, and of the fishermen's colonies and also of some nursery schools. This revealed that a much greater proportion of children in the poorer areas had a palpable liver, varying from one finger to three fingers (apparently healthy, and with no complaints). In contrast, among the children of fisherfolk (though belonging to the same economic grade of poor as the mill labourers), the proportion with palpable livers was much less. Some of these livers are fatty livers as revealed by the occasional liver biopsy we were able to make in such apparently healthy children. This survey seems to indicate that apart from the gross manifestation of protein deficiency like œdema, skin and hair changes, fatty infiltration of the liver without symptoms is going on in many apparently healthy children receiving low protein diets. The high incidence of cirrhosis liver among juveniles from the Madras General Hospital figures as compared to other similar hospitals in England and U.S.A. suggests that some of these children develop in later years clinical hepatic cirrhosis of the portal type. With the present increasing shortage of milk it is possible that these figures will go still higher in future years. The very good immediate response obtained by high oral proteid feeding supplemented by transfusions of blood and/or serum, and the unfortunate relapses that are only

too frequent after discharge, drive home a lesson of vital significance. Chronic malnutrition is a deflected life journey. Birth weight is often below normal, reflecting in many cases inherited malnutrition from the mother. Under the stimulus of adequate breast-feeding, the flight soars during its first compensatory phase and avoids the premature crash of malignant malnutrition as seen in bottle-fed babies. Soon after this the engine stalls; breast-feeding is prolonged, cow's milk is minimal and mixed feeding is delayed and relies too exclusively on 1 or 2 carbohydrates. At the same time in Madras, diarrhœal and dysenteric diseases operate and the life may crash into malignant malnutrition from which they die or recover into seemingly normal human adolescents and adults, but short in stature, weak in limb, sluggish in mind, low in hæmoglobin with a peculiar plasma protein mosaic and an abnormal liver and pancreas.

The possible solutions to the problem seem to be:—(1) Increasing the local production of cow's milk and proper distribution with the highest priority to children, whatever their economic means—this links up with larger, related issues such as the problems of fodder and cattlehealth, but they must be dealt with and solved.

(2) Supplementing the local available milk with imported milk powder and cheaper skim milk powder.

(3) Other mammalian milk like goats' and asses' milk may be utilised to children—this may not much increase the total available quantity of mammalian milk in a city like Madras, but in the rural areas might make an appreciable contribution to the problem. Propaganda is needed however to popularise goats' and asses' milk.

(4) Methods of transport of milk from distant places to the cities in a hot tropical country like India need to be improved utilising modern methods of pasteurisation, refrigeration and, if necessary, drying.

(5) Sources of proteid other than milk should be made available to the children. Meat and fish are at present not consumed as much as one would wish, because of the cost.

(6) Plant proteins, in view of the recent experiences of Dr. Dean of Cambridge among German children faced with milk shortage in post war years, hold out a bright hope. The proteins from various cereals locally available (along with soya bean flour) can be made into a powder form after the necessary transformation processes, care being taken to maintain the proper proportion, while the addition of a little skim powder enhances the beneficial effects of these plant protein mixtures. It is noteworthy that Dr. Dean was able to maintain health and promote normal growth among children by plant proteins alone without any milk.

It is very necessary in any scheme to ensure that the distributing agencies are properly planned out, and that the benefit of

protein diet, whether milk or substitutes, really reaches the children of the poorest classes. By a combination of some of these measures, perhaps we can ensure that children grow up uncrippled by such manifestations and that human wealth is not squandered by want and ignorance.

As regards treatment of the acute episode of nutritional œdema with or without its various concomitant accompaniments like dermatoses of various patterns, orogenital manifestations, anæmia, etc., we cannot overemphasize the importance of not wasting a lot of money and time on costly Liver Extract and Vitamin B injections. These have proved futile in our earlier series. We rely on high oral proteid feeding with half-cream lactic acid milk (since whole milk is not well tolerated in diarrhœic patients) skim milk powder, white of egg, meat, fish and wheat conjee, gradually building up the food tolerance. Blood and serum transfusions have proved invaluable and dramatic in the immediate response obtained while oral proteid is being built up. Casein hydrolysates are much costlier than plain naturally occurring proteid without proportionate benefit. We rarely use them and discourage its use by others both on account of the cost and because the doctor prescribing them often neglects to build up the diet, relying on the few grams of protein he supplies from the patent bottle or tin. Protein digestion and absorption is in our experience little impaired even in the worst cases, and there is no necessity for hydrolysing proteid. Trypsin was present in the 6 cases in which this was looked for by duodenal intubation.

We would like here to mention certain important byproducts arising from this study on fatty livers of nutritional dystrophy. One is the concept of death by acute hepatic parenchymal failure. We are familiar with this mode of death in acute fulminant infective hepatitis and in the so-called cholœmia. Also anyone who has dealt with these nutritional œdema cases will have had some dying suddenly and apparently inexplicably. The second is the use of concentrated serum and blood in diarrhœal disorders of infancy after the dehydration and chemical upset has been set right by Saline, Lactate and Potassium solutions. This seems to protect the liver and prevent death by acute hepatic parenchymal failure. The third point we would like to drive home is the distinction between fatty liver and infantile biliary cirrhosis. A lot of confusion exists in Madras and many a fatty liver of infants is misdiagnosed as early infantile biliary cirrhosis. The senior author with a research team has been making simultaneous clinical and liver biopsy studies of a series of children with enlarged livers. Out of this study emerges a fundamental pathological and, to a certain extent, clinical distinction between these two conditions both common in Madras. This question is gone into elsewhere fully in an article under publication. Fatty liver is due to dietetic proteid deficiency and responds often quickly to dietetic regime supplemented

by transfusions. Methionine and Choline aid recovery. On the other hand, in infantile biliary cirrhosis, the clinical picture, the naked eye and histological changes in the liver are different, there being necrobiotic changes in infantile biliary cirrhosis and minimal or no vacuolation. The evidence in our study is against this disease (infantile biliary cirrhosis) being primarily nutritional. We differ in this respect from other workers (Himsworth, Lahiri) who have commented on the possible nutritional etiology of infantile biliary cirrhosis. Arising from this and from extensive controlled therapeutic trials comes our third observation that Methionine and Choline do not have much influence on an infantile biliary cirrhosis liver.

The effects of protein deficiency studied in the acute form in children are no less applicable to other conditions in which such a state occurs. Protein depletion may result from a single or from multiple causes.

1. Insufficient intake of food, especially of protein.
2. Impaired absorption.
3. Excessive loss of protein in nephrosis, discharging wounds, acute illness following injuries and diarrhœal disorders.
4. Inability to utilise protein absorbed, as observed in extensive disease of the liver.
5. Increased metabolic states such as occur in thyrotoxicosis and leukemia.

Part of this work was done while making a study of chronic diarrhœal conditions under a grant from the Indian Council of Medical Research. The work on Infantile Biliary Cirrhosis which has been mentioned in comparison was made possible by a grant from the Madras State Medical Research Fund.

The authors are indebted to the Secretaries of the I. C. M. R. and M.S.M.R. Funds for permission to publish this paper.

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Action of Ascorbic Acid on Iron Metabolism

Non-corpuscular iron and vitamin C were first estimated in the blood of 5 healthy children. After oral administration of ascorbic acid, the iron content of the blood rose, the rise being greater when initial value was low. The increase was, however, temporary though when the value had been low it was subsequently maintained at a higher level than before. The effect was much *more marked after oral* than after parenteral administration and with sod. ascorbate than with ascorbic acid.

"The ascorbic acid and iron contents of the bloodstand in a definite relation to one another and ascorbic acid promotes the absorption of iron from the gastro-intestinal tract by forming a labile complex with it"—is the considered opinion of Dr. Bugyi of the Budapest Medical Hospital.—(Eng. Summary from *Paediat. d mub.*, Budapest, 6, 100).

DISEASES OF THE HEART IN CHILDHOOD

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DISEASES of the heart or cardiovascular system have come to constitute, in Western countries, the prime problem of the day; this attitude is justifiable, considering that heart disease constitutes, at the present time, the leading cause of death, even surpassing in its toll of life, the "dreaded trio" of tuberculosis, cancer and pneumonia[1].

Until quite recently, the subject of heart-disease in childhood had been neglected by the pædiatrician, partly due to the complexity of the problem of congenital malformations of the heart, partly to the lack of subjective manifestations in childhood and partly to a sort of "laissez-faire" attitude on the part of the medical profession itself. Thanks to the pioneer efforts of a few great individuals, the picture has changed somewhat completely, our present day knowledge of cardiology being in keeping with our knowledge of the other branches of pædiatrics.

Before embarking on the problem of heart-disease proper, it behoves us to realise that the normal heart of childhood differs in certain important respects from that of an adult. To enumerate but a few of these differences:

(1) The heart in childhood may lie, quite normally, on a higher plane than in adults, thus presenting the apex beat in the fourth instead of the fifth left interspace[2].

(2) The rate of pulsation of the heart is much more rapid in childhood, being reported as being (on an average) 130 per minute at birth, 110 at 2 years, 100 at 3 years, 90 at 8 years and 80 at 12 years of age (Hutchison and Monkrieff)[2].

(3) Sinus arrhythmia or the so-called "juvenile irregularity of the heart" (Mackenzie), with its phasic respiratory variations of pulse rate is physiological in childhood, being secondary to natural hypersensitivity of the vagus nerve mechanism; the old idea of enforcing bed-rest in any child with cardiac arrhythmia, howsoever benign, has been fortunately superseded, of late, by a more rational attitude of treatment based in accordance with the nature of the arrhythmia.

(4) The pulmonary second sound is relatively louder in children and may exceed in intensity the aortic second sound.

(5) Cardio-respiratory and functional or accidental murmurs are very common in children; in fact, it is almost customary to expect a soft and transient systolic bruit in a child after exercise or emotional excitement.

Classification of Diseases of the Heart

General interest in the ætiological incidence of heart disease has been growing steadily since the time of Cabot's[3] monumental paper in 1914; this is readily proved by the number of excellent statistical analysis, which have been annually pouring forth from different parts of the world. From the scientific point of view, the ætiological classification of heart-disease, recently proposed by the Criteria Committee of the American Heart Association[4], is certainly the best; it considers diseases of the heart under the following main heads *viz.*: (1) Congenital; (2) rheumatic; (3) syphilitic; (4) bacterial; (5) hypertensive; (6) arteriosclerotic (coronary); (7) pulmonary; (8) miscellaneous; and (9) "unknown." The relative incidence of these various ætiological forms varies considerably in different parts of the world. In Vakil's series[1] (1948) of 1,860 cases of organic heart disease, reported in India, the percentage frequency distribution was reported as follows:—*viz.*, congenital 1%, rheumatic 24·7%, syphilitic 12·9%, bacterial 1·1%, hypertensive-coronary group 33·2%, pulmonary 10%, miscellaneous 11·3% and "unknown" 5·7%. These figures, being inclusive of all ages, are not applicable to children, in whom "descrescent" forms of heart disease are rare and infections of the heart common.

Congenital Malformations of the Heart

In the countries of the West, congenital cardiac defects account for anything from 1 to 5 per cent of all cases of organic heart disease, the average being 2 per cent (White)[5]. In India, the corresponding incidence of congenital heart disease has worked out at 1 per cent (Vakil,[1] 1948); in childhood, the percentage frequency of congenital malformations of the heart works out at about 15 to 20 per cent and, in subjects under 5 years of age, the incidence of congenital lesions may exceed even that of rheumatic disease. According to White,[5] the relatively high incidence, in the tropics, of congenital heart disease, is attributable to a dearth of rheumatic infections; recent statistical analysis, however, from tropical and subtropical countries (Gunewardene[6]; Fernando[7]; Chavez[8]; Vakil[9]) question the validity of this old-fashioned belief in the rarity of rheumatic infections of the heart in hot climates.

The clinical complexity of congenital malformations of the heart has been responsible for the comparative neglect, on the part of the cardiologist and pædiatrician, of this important branch of medicine. Thanks to the pioneer efforts of Maude Abbott[10], Taussig[11], Muir and Brown[12] and the intensive application, in clinical practice, of newer methods of investigation such as cardiac catheterization and angiocardiography, we are now able not only to diagnose congenital heart disease with more conviction but to venture the exact nature of the malformation and to advise the correct form of treatment or surgical interference.

In spite of numerous attempts at classification of congenital heart diseases, there is no doubt that the clinical classification of Maude Abbot[10] remains to this day the most convenient classification in clinical practice. She divides congenital cardiac malformations, in accordance with the presence or otherwise of cyanosis, into three main groups, *viz*:—

(1) The acyanotic group, *i.e.*, cases without abnormal communications or shunts between the right and left sides of the heart. This group includes the entities of simple dextrocardia, coarctation of the aorta, aortic and subaortic stenosis, anomalies of septa and congenital heart block.

(2) The "potentially cyanotic" group or group of "cyanose tardive" *i.e.* cases of arterio-venous shunt with transient or terminal reversal of flow. This group includes patent ductus arteriosus, localised inter-auricular and inter-ventricular septal defects.

(3) The cyanotic group, "morbus cærulius" or "maladie bleue", with veno- arterial shunt. This includes cases of slight to moderate cyanosis such as cor triloculare biatriatum, pulmonary stenosis with patent foramen ovale, tricuspid stenosis and tricuspid atresia, cases of moderate to marked cyanosis such as Fallot's tetralogy and transposition of arterial trunks with ventricular septum defect, and cases of extreme cyanosis such as complete transposition of arterial trunks with intact ventricular septum, persistent truncus arteriosus, cor biloculare and certain types of pulmonary, mitral and tricuspid atresias.

Some types of congenital malformations of the heart, for instance auricular or ventricular septal defects, patent ductus arteriosus and coarctation of the aorta, are entirely symptomatic, being discovered usually on routine examination. In other types of congenital heart disease, any one or more of the following symptoms may be observed, *viz.* cyanosis, dyspnœa, cough, hæmoptysis, epistaxis, headaches, syncope or fainting attacks, convulsions, coma, paralysis, dysphagia, formications, precordial pains, etc.

The following is a brief account of some of the better known and clinically recognisable congenital heart lesions:—

Congenital dextrocardia or right-sided heart is clinically unimportant, being usually discovered accidentally on routine examination or fluoroscopy; it does not alter the life-span or curtail the activity of the patient in any way; two forms of this condition are, nowadays, recognised. The electrocardiographic findings, *viz.* inversion of all deflections of lead I and transposition of leads II and III are considered pathognomonic.

Coarctation of the aorta:—This implies a condition of stenosis, narrowing or localised constriction of the aorta at a point just distal to the origin of the left subclavian artery at or near the insertion of the ductus arteriosus. Two forms of coarctation are recognised,

after Bonnet[13], *viz.*, the "infantile" form and the much more common "adult" form. Coarctation of the aorta is fairly common in clinical practice and accounted for as many as 14·2 per cent of Abbot's[10] one thousand cases of congenital heart disease. Although, usually devoid of symptoms, this condition is recognisable on the following grounds *viz.*, (1) A high degree of discrepancy between the brachial and femoral blood pressure readings, the former frequently exceeding the latter by 100 mm. Hg. (systolic) or more. Brachial hypertension, in conjunction with a weak or absent femoral pulse and a low femoral blood pressure reading, should at once suggest the possibility of a coarctation ; (2) the presence of dilated, tortuous and pulsating vessels *viz.*, the internal mammary, intercostal, scapular and deep epigastric arteries, furnishes good evidence of the collateral circulation of coarctation ; (3) a loud and long systolic murmur, and a corresponding thrill are usual over the precordium back and anastomatic vessels ; (4) the demonstration of Roesler's[14] sign of notching of the lower borders of the ribs ; and (5) an enlarged cardiac silhouette together with a small or absent aortic "knuckle". The condition of coarctation of the aorta, once considered permanent and incurable, has been shown to respond favourably to surgical interference.

Aortic and subaortic stenosis :—In these defects, the presence or absence of symptoms will depend on the degree of stenosis and harsh systolic murmur with thrill in the aortic area and well conducted into the vessels of the neck, together with absent or diminished intensity of the second aortic second sound and an anacrotic, slow-rising or small pulse at the wrist.

Patent ductus arteriosus is a fairly common entity, ranking third in incidence in Abbott's[10] series of cases ; it shows a particular affinity for the female sex, is often asymptomatic and displays the following characteristic signs, *viz.*, (1) A cyanosis that is delayed, exertional or preterminal ; (2) a most characteristic, loud and continuous or loud and long systolic murmur (the so-called "machinery murmur"), heard best in the pulmonary area and frequently accompanied by a thrill ; (3) a characteristic prominence or convex projection, on fluoroscopy, in the region of the pulmonary artery coupled with a normal sized left auricle ; (4) sometimes, a high pulse pressure with or without a characteristic "water-hammer" pulse ; (5) a special predilection for bacterial invasion with the development of malignant endocarditis. Since the time of Gross and Hubbard[15] (1939), the condition has become readily amenable to surgery.

Auricular septal defects :—A defective auricular septum is the most common congenital cardiac abnormality. Symptoms often appear in early adult life. Cardiac enlargement is present, but there are no typical thrills or murmurs. Rœntgenology shows cardiac enlargement and a very prominent pulmonary conus ;

Electrocardiogram reveals right axis deviation. Patent interauricular septum may be associated with other abnormalities such as mitral stenosis (Lutembacher's disease[16]); auricular fibrillation is not infrequent.

Patent inter-ventricular septum:—(Maladie de Roger[17]) This is the commonest clinically recognisable congenital abnormality of the heart met with in practice. Diagnosis is mainly based on the presence of a loud systolic murmur best heard in the third and fourth left interspaces close to the sternum. A systolic thrill is often present. There are usually no symptoms. Congenital heart block may be an associated abnormality.

Pulmonary stenosis:—If the degree of stenosis is slight, cyanosis may be absent in childhood. Clubbing of fingers depends on the degree of cyanosis. Dyspnœa on exertion is a common symptom and paroxysmal attacks of nocturnal dyspnœa may precede development of cyanosis. Physical signs include a harsh systolic murmur and systolic thrill in the pulmonary area. The murmur is conducted towards the left clavicle. The pulmonary second sound is feeble or absent. Radioscopy shows a dilated pulmonary conus and the cardiogram, a right sided preponderance.

Tetralogy of Fallot[18]:—This consists of pulmonary stenosis associated with inter-ventricular septal defect, dextroposition of the aorta and hypertrophy of the right ventricle. It accounts for a majority of cyanotic congenital heart cases surviving beyond the age of 3. A systolic murmur may be heard in the pulmonary area, but is not as harsh as in pulmonary stenosis and does not tend to be conducted to the left clavicle. X-ray shows a boot-shaped heart, aorta projecting abnormally to the right and a marked concavity at the site of the pulmonary arc.

Rheumatic Heart Disease

The importance and high incidence of rheumatic fever and rheumatic heart diseases, although substantiated by a host of stastical analysis, are not sufficiently appreciated by the public. To give but a few instances, rheumatic fever accounts for 3 to 7 per cent of all medical admissions in the hospitals of Europe and America[19]; in the Scandinavian countries, the incidence of rheumatic fever works out at about 1 to 3 per thousand of the population[19]; in the U. S. A. there are said to be about one million cases of rheumatic heart disease[20]; about 25,000 deaths are reported annually from rheumatic heart disease in England and Wales, thus accounting for about 40% of all "cardiac deaths"[21].

The problem of rheumatic heart disease assumes an even greater magnitude in the case of children, in whom it ranks first in the causation of cardiac disability and death. Rheumatic fever displays a particular affinity for the younger age-groups; in Bach's[22]

opinion, nine out of ten cases of heart disease in childhood are of rheumatic ætiology; according to Keith[23], rheumatic heart disease has been detected in 0·1 to 2·08% of British and 0·9 to 1·36% of American school children. In a series of 2,500 school children of Bombay, investigated by one of us (R.J.V.) in 1939, the incidence of rheumatic involvement of the heart was found to be 2 per thousand children.

Although rheumatic heart disease has been considered rare or non-existent in tropical countries by one set of authorities (Rogers[24]; Cowan and Ritchie[25]; Clarke[26,27]), there is no doubt that present day authoritative opinion favours the opposite school of thought. It has been proved, quite convincingly by the statistical investigations of Stott[28] (1938), Chavez[6] (1942), Basu[29-31], Hughes and Yusuff[32], Hodge[33-35], Carruthers[36], Bannerjea[37], Kelly[38], Kutumbiah[39], Gunewardene[6], Raghavan[40], Fernando[7] and Vakil[9], that rheumatic heart disease is common enough in tropical and subtropical climates. In fact, 24·7% or "one in four" of all cases of organic heart disease studied by Vakil[1,9] (1948) were examples of rheumatic heart disease.

There is a tendency on the part of some clinicians to regard the rheumatism of childhood as clinically distinct from that of adults. Such an attitude is both unwise and unwarranted, despite the fact that in childhood rheumatic fever (1) does take a more insidious form; (2) starts with vague prodromal symptoms like pallor, fatigue, nervous irritability, myalgias, anorexia, vomiting, abdominal pains and epistaxis; (3) seldom displays the classical picture of an acute migrating polyarthritis with fever and toxæmia; (4) affects the heart with much greater frequency; and (5) exhibits other extra-articular syndromes like chorea, subcutaneous nodules and cutaneous rashes. In short, while the rheumatic infection of adults is dominated by an acute migrating polyarthritis, that of childhood is characterised by cardiac and other extra-articular syndromes.

The mode of onset, in children, is often insidious and preceded by an attack of septic tonsillitis, sore throat or scarlet fever, there being an intermediate latent period of one to four weeks. Several types of onset of infection are recognized in children depending on the presenting symptom or symptoms; arthritic, cardiac, choreiform abdominal, pyrexial, cutaneous, atypical and latent being some of the well-recognized modes of onset.

The incidence of carditis in rheumatic fever has been reported, in children under 12 years of age, by Poynton[41] (1925) as 61%, by Coombs[42] as 72·5% and by Coburn[43] as 65%. In view of such a high incidence of cardiac involvement, which incidentally is in the nature of a pancarditis, particular attention should be paid, in the case of rheumatic infection in childhood, to the early detection of heart involvement.

The following features are worthy of note in the early detection of *endocardial* or *valvular* involvement (endocarditis or valvulitis), *viz* :—

(1) A prolongation or muffling of the first heart sound at the apex, with loss of its distinctive muscular element (Keith[44], 1937). The altered first sound has been described as impure, muffled, murmurish or mushy (Friedberg[19], 1949).

(2) An early splitting or reduplication of the second heart sound at the apex, has been described by Sansom[45] and Cheadle[46].

(3) Blowing systolic murmurs, variable in intensity, location and propagation, are both frequent and early. Their distinction from the so-called functional or hæmic murmurs on the one hand and from organic murmurs of chronic valvulitis on the other, frequently taxes to the utmost the diagnostic ability of the physician.

(4) Bland, White and Jones (1935)[47] have reported the occasional early occurrence of a mid-diastolic apical bruit, which is transient, disappearing with the abatement of the rheumatic attack.

During the active phase of rheumatic infection, *pericardial* involvement, which may take the form either of acute fibrinous pericarditis or of pericardial effusion, is frequently overlooked, clinically. Although pericardial lesions are demonstrable in the post-mortem room, in practically all cases of rheumatic carditis, they are demonstrable clinically in only 25 to 35% of cases (Garrod[48]); in other words, unless the involvement of the pericardial layers is severe and extensive, the condition may remain clinically silent. The following symptoms and signs are of value, however, in the early recognition of pericarditis, *viz*: (1) precordial pain or sense of oppression or fulness; (2) sudden development of restlessness, vomiting, dyspnœa, cyanosis or cough; (3) a sudden rise of temperature or pulse rate; and (4) the appearance of a friction rub.

Myocardial lesions, although often difficult of clinical recognition, are very common and of the utmost importance. The development of a persistent or 'disproportionate' tachycardia, *i.e.* high pulse rate in relation to the temperature, or a high 'sleeping pulse rate' (Sclesinger's sign), a sudden unexplained rise of temperature in the course of rheumatic fever, a sudden alteration in quality, intensity or pitch of the first heart sound at the apex (distant, muffled or valvular type of sound), a tic-tac rhythm or embryocardia, a feeble and diffuse apex beat, a blowing systolic murmur, development of cardiac failure (left or right-sided) or a disturbance of cardiac rhythm should suggest the possibility of a myocardial involvement.

Syphilitic heart disease or cardiovascular syphilis :—Although syphilis is regarded as 'the second most common and important cause of infectious cardiovascular disease', it accounts for a very small percentage of cases of heart disease in childhood, its highest

age incidence being in the forties. In White and Jones' (1928)[49] series of 95 cases of cardiovascular syphilis, there was only one case under 20 years of age.

Congenital syphilis rarely leads to cardiovascular disease; although the causative organism of syphilis is frequently detected at postmortem, its mere presence does not spell cardiovascular syphilis. In McCulloch's series[50] (1930) of 498 children with congenital syphilis over two years of age, only 5 showed signs of cardiovascular syphilis.

Bacterial endocarditis also known as septic, infective, malignant or ulcerative endocarditis, respects no age or sex. Its acute and subacute forms are distinguished on the basis of the duration and course of the disease and also on the nature of the infecting organism or organisms. Although cases of bacterial endocarditis are usually found in adult life, they do occur in children. It is only within the last few years, thanks to the newly discovered chemotherapeutic and anti-biotic agents, that the invariably fatal outlook of this disease has been virtually reversed; with intensive treatment, correctly applied, it is now possible to cure the great majority of cases.

In the *acute* form of the disease, the causative organism, which may be a streptococcus, hæmolyticus, staphylococcus aureus, bacillus coli, pneumococcus, meningococcus or gonococcus, invades the heart secondarily in the course of some acute or serious illness like pneumonia, puerperal sepsis or pyæmia. The condition is characterized by a high and swinging temperature, intense toxæmia, frequent chills or rigors, drenching sweats, prostration, delirium, progressive pallor or anæmia and embolic phenomena; involvement of the heart is usually suggested by the appearance, accentuation or modificaton of a murmur or murmurs.

The *subacute* form, which in 90 to 95 per cent of cases is due to a streptococcus viridans or non-hæmolyticus, invading valves which are chronically affected by rheumatic endocarditis or congenital anomalies, accounts for about 1 to 2 per cent of all heart cases. It is commonest between the ages of 15 and 30 years (White [5]). In Kelson and White's[51] series (1944) of 250 cases of bacterial endocarditis, 6 were under 10 years of age and 42 were aged 10 to 20 years. Goetsch[52] has reported the disease in a child aged 1½ years. The clinical picture of subacute bacterial endocarditis presents many interesting facts, *viz*: (1) the symptoms and signs of an infective process, such as fever, chills, sweats, malaise, loss of weight, appetite and strength; (2) embolic phenomena, leading to infarcts of spleen, kidney, brain etc.; (3) Osler's nodes[53]; (4) clubbing of nails; (5) hæmorrhages into the skin, epistaxis, hæmaturia etc.; (6) characteristic pallor and "cafe au lait" appearance (Libman[54]); (7) splenomegaly; (8) symptoms and signs of cardiac involvement; (9) leucocytosis; (10) positive blood cultures; and (11) red blood cells in the urine.

Other infections of the heart:—In the acute stage of *diphtheria*, although the pericardium and endocardium are hardly ever involved (Sutherland and Willis[55]), grave myocardial damage or myocarditis is fairly common and carries with it a very grave prognosis. Symptoms and signs suggestive of myocardial involvement in diphtheria are many and varied *viz.*, dyspnœa, precordial oppression, palpitation, abdominal pain, vomiting, lassitude, prostration, restlessness, cough, pallor, cyanosis, tachycardia, bradycardia, cardiac arrhythmia, gallop rhythm, displacement of apex beat, apical systolic murmur, moist sounds in the chest, heart-block and sudden death.

In spite of the frequency of myocardial involvement, chronic heart disease from diphtheria, unlike rheumatism and syphilis, is extremely rare (Jones and White[56], 1927; Alstead[57], 1933).

Treatment of diphtheritic myocarditis consists of the prompt administration of massive doses of antitoxin, enforcement of complete and prolonged bed-rest and intravenous glucose therapy.

Fiedler's myocarditis is an acute interstitial myocarditis of unknown ætiology. The onset is insidious or acute, fever is common and a rapidly progressive congestive cardiac failure dominates the clinical picture. Embolic phenomena may be observed in the absence of positive blood-cultures. The prognosis of this rare disease is very grave, a fatal outcome being customary in a matter of weeks or months.

Myocardial lesions, like those in diphtheria, may be encountered in other infections like measles, pertussis, typhoid, pneumonia and even acute anterior poliomyelitis.

Hypertensive heart disease.—Taking into consideration all ages, there are no two opinions about hypertensive heart disease being the commonest and the most important form of organic heart disease the world over. In Bombay, it is said to account for 29 per cent or almost one-third of all cases of organic heart disease (Vakil,[58] 1947). In spite of this high overall incidence, hypertensive heart disease plays but a small part in the genesis of cardiac disease in childhood. Out of 708 cases of hypertensive heart disease, reported by White and Jones[49] (1928), only 0·5 per cent of the cases were below the age of 20 years. In Vakil's[58] series (1948) of 538 similar cases, observed in India, 3 cases (or 0·6%) were in the first decade of life and 20 cases (or 4%) in the second. The youngest case of this type, on record, is that of a two-year old boy, reported by Taussig and Remsen[59] (1935).

Hypertension, when encountered in children, is seldom of the type commonly referred to as essential or primary or genuine. Unlike grown-ups, children are usually affected by hypertension that is secondary either to coarctation of the aorta, polycystic disease of the kidneys, chronic stage of diffuse glomerulonephritis, Cushing's basophilic syndrome or suprarenal tumours. When

essential hypertension does overtake a child, it is usually of fulminating type, the so-called 'malignant form' of hypertension (Volhard and Fahr).

Coronary heart disease:—Although no age-group, 'from youth to extreme old age' is considered immune from coronary heart disease, clinically recognisable coronary disease is rare in subjects under 20 years. In White and Jones' [49] (1928) series of 864 cases of coronary disease, White, Bland and Miskall's [5] (1943) series of 497 cases of angina pectoris and Bland and White's [5] (1936) series of 461 cases of coronary thrombosis, not a single case was observed under the age of 15 years. In Vakil's[60] series (1948) of 250 cases of coronary disease, studied in India, there were 3 cases under 19 years of age, the youngest being a girl of $8^1/_2$ years with a history of anginal attacks and an electrocardiographic picture of posterior wall infarction.

For practical purposes, however, it is unwise to diagnose angina pectoris or coronary occlusion in a child, unless and until there is indisputable evidence to support such a diagnosis. From the pathological standpoint, it is difficult to understand why clinical coronary disease is so rare in childhood, considering that definite lesions of coronary atherosclerosis are not unusual, according to Wolkoff (1929),[5] Ehrich [5] (1931) and Leary [5] (1935), even during the first decade of life.

Pulmonary heart disease or cor pulmonale:—Although of recent recognition, this variety of heart disease is important, considering that it accounts for 6·8% (Scott,[61] 1941) or 10% (Vakil,[1] 1948) of all cases of heart disease. In childhood, the incidence is very much less, the acute form of cor pulmonale (McGinn and White,[62] 1935) secondary to massive pulmonary embolism being practically unknown. Even the chronic form, *i.e.*, secondary to chronic lung disease, or severe deformities of chest, is very rare in childhood, the youngest case of this type, encountered by White,[5] being a child of 11 years. In childhood, chronic cor pulmonale when encountered, usually belongs to the group of primary pulmonary endarteritis.

Cardiac arrhythmias:—Alterations of cardiac rhythm are most uncommon in children and are more often physiological or without any clinical evidence of organic heart disease.

Paroxysmal tachycardia:—It is known to occur in infants and may start soon after birth but the incidence is more common in older children. In infants the attack may start as a convulsion or fainting, or the infant may become restless, vomit and may become dyspnœic and cyanosed. Older children might complain of epigastric or precordial pain, palpitation and dyspeptic symptoms. Cardiac failure may set in if the attack is prolonged and the liver may enlarge considerably.

Extrasystoles:—Premature contractions readily arise from ectopic foci of heart muscle in children. They have been recorded in

the fœtal heart and are fairly common in infants. In a majority of cases the heart is normal. Among the varied causes, may be constipation, worms, infections like tonsillitis or the onset of fevers.

Heart block:—Prolongation of P-R interval is an important and sometimes the only indication of involvement of the myocardium in acute rheumatic fever. In diphtheria the degree of block may vary from mere prolongation of P-R interval to complete heart block. Congenital heart block may be associated with patent interventricular septum.

Auricular fibrillation:—One of the common cardiac irregularities in adults, it is very rare in children. It may seldom occur during the course of acute rheumatic fever but is perhaps more common in diphtheritic myocarditis. Paroxysmal auricular fibrillation may occur without apparent cause.

Auricular flutter may be met with in a paroxysmal form in infants. It is known to occur in diphtheria.

Anaemias:—Severe chronic anæmia in a child may give rise to dyspnœa on exertion, precordial pain, palpitation, œdema of feet, cardiac enlargement and a soft systolic murmur. Heart failure is rare but may complicate severe anæmia. Sickle cell anæmia is well known to produce cardiac symptoms, or symptoms resembling rheumatic fever and heart disease.

Avitaminosis:—Among the diseases resulting from deficiency of vitamins, affection of the heart is a prominent feature in beriberi. The infantile form is practically confined to breast-fed children of mothers who are themselves suffering from vitamin deficiency. In the severe or acute type, the child becomes œdematous, develops convulsions and succumbs to a rapidly developing cardiac failure. In the moderately severe or less acute type, there is a disinclination for feeds, restlessness, crying due to abdominal colic and palpable tender liver. Tachycardia, hurried respirations, cyanosis and generalised œdema with enlargement of heart are soon noted. Signs of increased intracranial pressure and aphonia may precede death. In the chronic type, there is anorexia, progressive weakness and breathlessness. The heart is very much enlarged. Therapeutic test with vitamin B helps to confirm the diagnosis.

Endocrine and metabolic hyperthyroidism may give rise to palpitation, tachycardia, dyspnœa, cardiac enlargement and even failure.

Cretinism and juvenile myxœdema:—Hypothyroidism in infancy and childhood will cause retardation of growth, mental deficiency etc., but cardiac manifestations like bradycardia and enlargement of heart are very rare.

Glycogen disease:—In this metabolic disease, there is cardiac enlargement without obvious cause. Diagnosis may be confirmed by presence of hepatomegaly, fasting hypoglycemia and failure of rise of blood sugar following injection of Adrenalin.

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INFANTILE BERIBERI

Human Milk Intoxication Due to B Avitaminosis

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Introduction.—Infantile beriberi, as the name implies, is a disease of the infants, who have been, at some time, breast-fed. It is a separate entity, differing considerably from the adult types of beriberi in its ætiology, symptomatology and course. It is a "great imitator", for even without the usual secondary infections, it may imitate the diseases of the brain, respiratory tract, heart, kidneys, intestinal tract as well as a number of nutritional diseases. The differential diagnosis of infantile beriberi with secondary infections includes almost all the known diseases.

Infantile beriberi was first described by Hirota in 1891. The disease differs from the adult type, specially in its acuteness and suddenness. Apparently healthy infants may succumb to it within a few minutes. The mother may make the simple but dramatic statement that one or more of her infants has "turned blue, sighed and died". This acuteness gave rise to the assumption that the condition is due to an intoxication—human milk intoxication. That it is an intoxication has been amply justified by recent investigations.

The disease is very common in the Phillipines, China and Japan. It is one of the most important causes of infant mortality in these countries. The occurrence of infantile beriberi in India has been recorded only recently, though the adult form has been known for a long time. Aykroyd and Krishnan (1941) reported cases of infantile beriberi in the Northern Circars of the Madras Presidency where beriberi in adults is very common. Krishnan, Ramachandran and Sadhu (1945) have confirmed that infantile beriberi is common in that part of the country and that it is an important public health problem for India. It is also quite possible that this disease is also common in other parts of India where adult beriberi is encountered, but most probably it is not so well recognised because of its multiple and diverse features and its extremely short duration.

Aetiology.—In the absence of thiamin, carbohydrate cannot be properly oxidised to its end-products. In the utilisation of carbohydrate, thiamin takes part in an enzymic system, known as Carboxylase. This enzyme is specially needed for the utilisation of Pyruvic Acid, which is a normal intermediate product of carbohydrate metabolism. In thiamin deficiency, this product accumulates in the system due to non-utilisation and, associated with this, other intermediate products also accumulate. Some of these intermediate products are Lactic Acid, Acetoacetic Acid, Glyceraldehyde, Dihydroxy Acetone, Methyl Glyoxal etc. Methyl Glyoxal which

has proved to be toxic has never been isolated from the breast milk of healthy women (Kermak, Lambie and Slatter, 1927 and Vogt-Moller, 1931). Pyruvic Acid and Pyruvates are also toxic. Besides the above two, other intermediate metabolites, in the quantity present in the milk, may take part in the causation of intoxication (Haynes and Weiss, 1940). Their action most probably is synergic and cumulative. All these substances are present in the human milk of B_1 avitaminotic women (Takamatsu, 1934). In fact, the accumulation of these substances in the human milk changes the taste of milk in such a way that the hungry infant refuses to take the milk. It is suggested that the human milk containing such intermediate products is the cause of infantile beriberi or human milk intoxication.

After ingesting these intermediate products, the infants try to excrete or oxidise them. Oxidation of these products is only possible, if sufficient amount of thiamin is available, whereas in, actual it is just the opposite. Probably, every infant has a store of thiamin, even if it be very small, and therefore the toxic action is delayed. It is however immediate, if the infant is already avitaminotic.

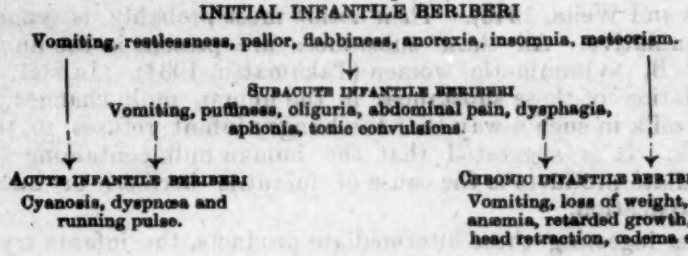
Clinical features.—The acuteness of symptoms of infantile beriberi will depend on the amount of milk ingested and hence acute infantile beriberi is common among well-fed babies.

It is common between the second and the sixth month of life. The clinical picture of the disease varies according to the organs and tissues where there is greatest concentration of these toxic metabolites (Fehily, 1941). This explains many of the contradictory statements made by authors on infantile beriberi and also explains why this disease is known as a great imitator. According to Fehily, this also explains the rather constant enlargement of the right ventricle, since this part of the heart is the first to receive the blood from the digestive organs, still rich in toxic metabolites. The symptoms of infantile beriberi may be divided, according to acuteness, into symptoms due to intoxication and symptoms due to avitaminosis. The symptoms of intoxication, such as vomiting, restlessness, abdominal pain, cyanosis, dyspnœa, running pulse etc., prevail in the initial, subacute and acute forms of infantile beriberi whereas chronic infantile beriberi is dominated by symptoms of B_1 avitaminosis such as anorexia, retarded growth, loss of weight, constipation etc. As the last mentioned symptoms are encountered also in infants who have never been breast-fed but who suffer from B_1 hypo- or avitaminosis, these conditions may give rise to symptoms simulating infantile beriberi. These conditions in artificially fed infants are often diagnosed as malnutrition but in contrast to chronic infantile beriberi, symptoms such as aphonia, head retraction are not encountered in the B_1 hypo- or avitaminosis.

After cessation of breast feeding, the symptoms of intoxication promptly disappear, whereas those of avitaminosis persist for weeks

or months, specially if the substitute foods are deficient in thiamin or its amount is insufficient to remedy the existing deficiency.

Infantile beriberi can be thus broadly divided into the following types according to the signs and symptoms:



In all stages of infantile beriberi, secondary infarction mainly of the respiratory tract is very common. Bronchitis and bronchopneumonia with accompanying fever is more common. In uncomplicated cases of infantile beriberi, temperature is normal and even in some cases subnormal.

PATHOLOGY.—Nothing much is known about the pathology of this condition. The most characteristic feature about the autopsy report is the absence of any pathological finding to account for the death in uncomplicated cases. External œdema is very unusual in infantile beriberi but internal anasarca is usually encountered. This anasarca includes not only the pleural, peritoneal and pericardial effusions but there is also œdema of the heart, brain, lungs, kidney, spleen etc. One characteristic thing about the enlargement of the right ventricle of the heart in these cases is its rapid diminution to its original size if the vitamin therapy is started early and effectively. This also suggests that this enlargement is due most probably to interstitial œdema.

DIAGNOSIS.—It is clear from the above that it is rather difficult to establish the cause of death in infantile beriberi either from the clinical features or from the post-mortem report.

Unlike adult beriberi, there is not the proper clinical diagnosis nor the helpful history of the illness prior to death. In addition, the fact that the child is usually of well-nourished type, without any outward sign of the disease, is very confusing and has frequently given rise to the suggestion of foul play or poisoning.

The manifestation of infantile beriberi is so diverse and multiple and secondary infections are so common, that there is every possibility of confusing the disease with almost any of the known diseases. Fehily, while working in Hongkong, has stated that the following diseases can easily be confused with the following types of infantile beriberi:

Initial infantile beriberi.—1. *Overfeeding* :—Many of the signs and symptoms of overfeeding are similar to this type of infantile beriberi. The medical history of other children (living or dead), mother's diet during pregnancy and lactation and symptoms of avitaminosis confirm the diagnosis.

2. *Status lymphaticus* :—Absence of enlarged tonsils, palpable lymphatic glands, enlarged thymus, together with the clinical and dietetic history of the mother confirm the diagnosis.

3. *Bronchitis* :—Hoarseness and aphonia in infantile beriberi may by mistake be attributed to that caused by the strain from crying or suspected laryngitis. The enlarged right ventricle, the maternal diet and the avitaminotic symptoms of the mother would confirm the diagnosis. The aphonia in infantile beriberi is due to œdema of the larynx (even requiring tracheotomy) and the degeneration of the recurrent laryngeal nerve. The aphonia is very characteristic and is described as the so called "visible cry". The mouth is open, there are crying grimaces but no sound is heard.

Subacute infantile beriberi.—1. Dyspepsia :—The diet of the mother and the signs of avitaminosis help in the diagnosis. 2. Meningitis :—Normal pressure and the clarity of the cerebrospinal fluid, absence of the bulging fontanelles and Kernig's sign indicate infantile beriberi. In addition, the diet and history of the mother also helps in the diagnosis. 3. Nephritis :—Administration of thiamin is followed by profuse urination and considerable loss of weight due to reduction of œdema. 4. Peritonitis :—Enlarged right ventricle and the œdema help in the differentiation. 5. Helminthiasis :—History is helpful. 6. Tetany. 7. Cerebral injury. 8. Diphtheritic paralysis.

Subacute infantile beriberi.—1. Bronchopneumonia :—This is one of the most frequent complications of infantile beriberi. In a certain percentage of cases, pulmonary œdema is encountered. The above two factors mask the real picture making the diagnosis almost impossible. 2. Cardiac diseases. 3. Laryngeal diphtheria. 4. Accidental poisoning. 5. Laryngismus stridulus.

Chronic infantile beriberi.—1. Malnutrition. 2. Tuberculosis. 3. Syphilis.

Krishnan, Ramachandran and Sadhu (1945) are of the opinion that therapeutic test is of great importance in the diagnosis of infantile beriberi. They have stated that the injection of 5 mgm. of Thiamin brings about a dramatic and remarkable effect on the infants. The screaming, paroxysm and the general appearance improve greatly.

TREATMENT :—The treatment of infantile beriberi consists of the following :—(1) Immediate stoppage of the breast milk. (2) Injection of large doses of Thiamin to the infant. Foreign workers give upto 50 mgm. of Thiamin in severe and moribund cases. (3) Improvement of the diet of the mother.

Public health problem.—Infantile beriberi or human milk intoxication is a public health problem in India as in other "beriberi areas" of the Orient, but unfortunately, it has not been given the right place due most probably to the difficulty in its diagnosis and the extremely short duration. It is also worthwhile to remember that it differs from all other deficiency diseases in the fact that it is primarily an intoxication in the infant caused by the deficiency of Thiamin in the mother. Steps must be taken in the right direction, so that the condition is widely recognised by the medical profession in India.

In this connection, it has to be remembered that this disease is caused by taking human milk, which is regarded as the best food for the infant. Fehily (1944) has aptly remarked—"the statement that the breast milk is the best food for the infant should be qualified by the proviso that if it is secreted by the healthy mother". The truth and the magnitude of the above statement can be verified by an interesting case which she has seen in Hongkong. She encountered a case of acute milk intoxication in a 5 month old poorly nourished Chinese infant, whose mother admitted, on close questioning, that until a week previously, the child had been fed on sweetened condensed milk, she being a professional wet-nurse. During these five months, she nursed two infants, both of whom succumbed to a disease similar to that of her own child.

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Rheumatic Fever and Tonsils

Tonsillectomy is indicated in all forms of rheumatic fever if the patient has repeatedly suffered from acute tonsillitis or if there is a chronic inflammation of the tonsils. However, in similar cases other infectious foci should be also eliminated. In case of focal infection the patient should not be operated on before acute symptoms have disappeared. Early tonsillectomy may involve complications in labile allergic states. The bacterial flora of the normal pharynx is fairly constant, and there are no relevant changes in the tonsils of children suffering from rheumatic disease. Prophylactic tonsillectomy does not significantly inhibit the involvement of joints. Similarly, tonsillectomy following an acute rheumatic infection has very little, if any, prophylactic effect on the frequency of relapses.—(E. Gyorgy, *Paediat. danub.*, 5 : 340 (June) 1949.—*American Journal of Diseases of Children*).

INFANTILE CIRRHOSIS

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THE fundamental one should grasp is that infantile cirrhosis is in no way a separate entity from cirrhosis occurring in adults, as far as its etiology, course and prognosis is concerned. This condition has gained its notoriety due to its prevalence in the tropics, to age incidence and frequent malignant outcome.

Infantile liver has worked up the minds of the public who regard it with great awe as, till of late, nothing was known about its scientific treatment, the etiology being only a matter of guess work and the outcome fatal.

This gave a strong foundation to mystic liver cures and various so-called infallible remedies in other systems of medical practice.

Now it is regarded as a deficiency disease due to deficiency of certain essential aminoacids which first lead to fatty degeneration and which, if of long standing, lead to fibrosis.

Infantile liver may include a number of conditions :—(1) Functional disorders. (2) Multilobular cirrhosis. (3) Chronic biliary cirrhosis. (4) Chronic infective hepatitis. (5) Syphilitic cirrhosis. (6) Banti's disease. (7) Hepato-lenticular degeneration.

1. *Functional disorders* :—Chronic or recurrent symptoms of digestive upset furring of the tongue, foul breath, and lack of appetite, low unaccountable temperature are the chief symptoms of this condition. If these symptoms are recognised early and the underlying liver condition treated the disease process can be completely reversed and ultimate prognosis is excellent. This, if neglected, well lead to cirrhosis of liver with its alteration of size.

2. *Multilobular cirrhosis* is the commonest variety. Age incidence from infancy to twelve years of life. "It is just like alcoholic cirrhosis of adults," was well said by Hutchinson in one of his lectures—a remark which has stood the test of time.

Thanks to Himsworth, Glynn, Gillman and others, we know that alcoholic cirrhosis is not due to any toxins like alcohol etc., but due to deficiency of essential aminoacids specially Choline and Methionine and the same stands true for infantile cirrhosis.

In India this disease is more common among the children of vegetarian families, especially the Banias, the Gonars and the poor, who cannot afford to take enough of protein in their diet.

This condition is very common among the South African children, a class who take a very highly spiced diet causing chronic

gastro-enteritis which prevents the absorption of sparingly present aminoacids. The staple diet being maize, vegetables, fermented milk and sometimes beans.

General food shortage, specially wheat, and poverty tend to increase the incidence, so do the subsequent pregnancies.

The deficiency may be either congenital or acquired as in deficient quality of mother's milk, over-diluted milk, too fatty diet, *e.g.*, vegetable ghee, cooked food and other environmental conditions.

This is a preventable malady which can be prevented by giving adequate proteins and other essential factors of diet to the mother and in early infancy. As everywhere else there is no cure of fibrosis although the process can be checked and liver cells helped in their enormous capacity to regenerate some of these cases show a weak positive aldehyde and Chopra test and this reaction should be interpreted with care in a cirrhotic child.

3. *Syphilitic cirrhosis* :—A positive parental history is often elicited in such cases. Liver and spleen are considerably enlarged while ascites is not a common feature. The child only in some cases may show other stigmata of syphilis. Positive blood serum reaction in the way of W. R. and Kahn test is diagnostic. This condition is more often diagnosed than really exists.

4. *Biliary cirrhosis* is secondary to obstruction to bile ducts which are usually congenital and manifested early in infancy. Jaundice is a marked feature. The treatment is surgical.

5. *Chronic infective hepatitis* :—This condition is being recognised more often, usually there is a history of preceding acute attack of infective hepatitis which, unlike an average case, is very slow to recover. Rarely the acute attack passes unobserved. The real cause may be conditioned protein deficiency.

6. *Banti's disease* :—This again is one of the rare diseases of childhood. Repeated hæmatemesis and slow progress of the disease are the clinical criteria to differentiate it from portal cirrhosis. Splenectomy is of no use.

7. *Hepato-lenticular degeneration* :—This is the rarest form in which infantile cirrhosis may exist. The nervous signs and symptoms of the affection of extra pyramidal tract is the presenting symptom. Liver is also found enlarged and cirrhotic course is prolonged.

PROGNOSIS.—1. If diagnosed early in the pre-cirrhotic stage the prognosis is excellent.

2. In the early cases when there is only a moderate enlargement of liver and it is not hard, prognosis is still very good.

3. When there is slight jaundice and low temperature prognosis is still hopeful.

4. If the liver is more than four finger breath, there is deep jaundice or high unaccountable temperature, or abdomen is distended with free fluid, the prognosis is invariably fatal.

5. Hæmorrhages and diarrhœa are also very grave symptoms. Any secondary infection as tuberculosis, pneumonia etc. of the lung is the common terminal event.

TREATMENT.—*Diet* :—High protein, average carbohydrate and low fat are recommended in such cases.

Prophylactic :—Mother, and later the child, should be given a proper balanced diet.

Curative :—Supplying all essential aminoacids supplemented with proteolysed Liver Extract Vitamin B complex and Vitamin C by mouth or parenteral, depending on the state of the gastro-intestinal tract.

Diarrhœa and intercurrent infections should be promptly checked by chemotherapeutic drugs.

Vitamin K should be given when prothrombin index is lowered or there are bleeding tendencies.

Intraheptal and I. V. Protein Hydrolysate are also useful before stage IV.

Cholagogues :—Like bile salts, mercury, salines, salicylates and bitters should be used to keep the flow of bile satisfactory and with the hope of relieving congestion from the liver.

Extract Kalmeg Liq. has been also used with indifferent results.

Ventriculin therapy has worked wonders in the hands of some workers in South Africa and should be given a trial in this country.

Lambliasis in Childhood

The author, after a critical examination of the question of the pathogenic importance of Giardia lamblia, reports on 250 clinical cases of lambliasis observed in the last five years and on the remarkable diffusion of Giardia in the province of Messina and in Calabria.

The picture most frequently seen is that of enterocolitis dysenteriform ; the course is sometimes acute, most frequently subacute or chronic. There are not infrequently cases of dyspepsia, accompanied sometimes in nurslings with dystrophy.

The author mentions some clinical pictures less frequently seen, and he illustrates some cases of Herter's disease (intestinal infantilism), toxicosis, hepatobiliary syndrome, pseudo-tuberculous syndrome, hypochromic anæmia, dermatitis, pellagra syndrome and allergic syndrome. The therapy of choice is acridinic treatment supplemented in the chronic forms by arsenical treatment.—S. Barberi, *Ann. ital. di pediatric*, 1:16 (Dec.) 1948—*American Journal of Diseases of Children.*

CONGENITAL (INHERITED) SYPHILIS

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Incidence.—The official figures for congenital syphilis in the Indian Sub-Continent, whether they be attending the clinics for the first time or the recorded deaths from the disease, fail to give an adequate picture of the evil. There is, however, no denying the fact that the disease is widely spread to invite our attention. During the last four decades, however, after Ehrlich found arsenic as an almost specific treatment for syphilis, there is a profound change in the way in which the disease manifests itself as compared to the cases seen in the early years of the present century.

During my student days and for some years afterwards frank cases of the disease were very common and their recognition even before the discovery of Treponema Pallidum and Wassermann reaction presented no difficulty. The snuffles, the characteristic and rather pleomorphic rash on the face and body, particulary in the ano-genital region, the enlargement of the spleen and liver, the lymphadenitis and the lesions of the bones and joints rendered most of the cases obvious. The child may give the appearance of a dwarf or cretin, but thyroid etc., have no beneficial effect.

Where the parents are syphilitics, pregnancy does not usually continue to full term, the mother miscarrying perhaps at the end of six or seven months. In these cases the child may be well formed and live independently for a little while, but not infrequently it is still-born and in many cases macerated; in these cases the tissues are often teeming with spirochætes. Miscarriage may occur for several pregnancies and, then, a living child, be produced. If the infection occurs early in the pregnancy the mother usually goes to a full term, but her offspring is born with all the signs of congenital syphilis. If the disease occurs late in the pregnancy, the fœtus may escape transplacental infection but becomes inoculated through a small abrasion acquired during the process of labour. In other instances, however, a living child may be born at full term at the end of the first pregnancy inspite of the syphilitic infection of the parents. This child may show evidence of the disease at birth, but more frequently appears to be healthy, specific manifestations not showing themselves for some weeks. The infectivity of the mother and the degree of virulence of the disease in successive children decrease with time and it is possible that after five or six years of untreated syphilis, a mother may bear a healthy and untainted child; although infection has been known to be transmitted much later. After the first year in untreated or insufficiently treated cases any of the tertiary phenomenon which appears in acquired syphilis may develop, but in addition to these, peculiar manifestations enumerated below may be produced.

The victims of congenital syphilis after ten or twelve years of age may present a facies which is characteristic—an over-hanging forehead, perhaps frontal bosses, a depressed nasal bridge, striated scars radiating from the corner and other parts of the lips, with a sallow, earthy complexion. On closer observation of the eyes and teeth one may detect the opacities of old keratitis and the changes in the upper incisors which are claimed by Hutchinson to be pathognomonic. These teeth are widely gapped, irregular and so deficient in enamel over the anterior and median parts of their cutting edges that the resulting crescentic notch gives them a striking appearance. Such facies accompany deafness, mental deficiency, physical infantalism, tibial deformities and chronic arthritis especially of the knee joints. The diagnosis may be clinched if the blood gives positive Wassermann reaction.

Congenital syphilis is a potent cause of marasmus. Many congenital syphilitic children without any of the better known evidence of syphilis fail from simple inability to thrive, although born fine, healthy looking infants, presently waste and pine and they may be said rather cease to live than in the ordinary sense to die of a disease.

DIAGNOSIS.—The diagnosis may be obvious from snuffles, skin lesions, parrot's nodes, condylomata. Congenital syphilis may be from known parents or detected from Wassermann or Kahn's serum test. It may be suggested by a knowledge of the family history, but in many cases it will be a diagnosis of surmise only unless confirmed by serological results.

Occurrence:—There is perhaps no tissue of the body which is not affected by Treponema Pallidum. It is therefore essential to study it as it produces its effects on various systems of the human body. Let me therefore detail each system separately and enumerate the effects of congenital syphilis on it.

Skeletal system:—Congenital syphilitic epiphysitis appears earlier than in rickets and scurvy and sometimes can be demonstrated radiographically as early as the second month. The bones of the knee and wrist joint are the commonest to show the characteristic changes and the pain of the lesion is such that the affected limb is held quite still (syphilitic pseudo-paralysis). The radiograph shows broadening and irregularity of the metaphysis which is quite different from rickets in that the outline, though irregular, is dense and sclerosis is predominant. Typically, the layer of dense irregular bone cupping the metaphysis is bounded on the shaft side by a thin layer appearing translucent in the X-ray plate, while the cortical region of this part of the bone shows pinched out areas of sub-periosteal erosion. Other bone manifestations present in syphilis of infancy include, areas of periosteal new-bone formation, syphilitic dactylitis and also parrot-nodes. A tenderness of the cranial bones

is seen in congenital syphilis and is caused by the rarefying (craniotabes) or hyperplastic (hot-cross-bun skull) osteitis present. These last are bosses on the bones of the vertex of the skull which result in hot-cross-bun head, often of more exaggerated shape than in rickets.

Syphilitic dactylitis occurs at an earlier age than tuberculosis, usually before twelve months with other signs of syphilis (skin rashes, snuffles, thick mop of coarse hair, oral fissures etc.) and the Wassermann reaction of the infant and parents will in most cases aid the diagnosis. Locally the distinction is difficult, there is not the same tendency in syphilis to erosion of the bone, or the formation of sinuses and the new bone is usually thicker and denser, but these slight differences are unreliable.

Syphilitic periostitis:—At about the age of nine or ten years congenital syphilitics are liable to local or diffuse deposition of dense periosteal bone. This typically occurs in tibia which also undergoes some elongation resulting in the well known Sabre-shape. Other signs of congenital syphilis appear at this age including Clutton's joints, interstitial keratitis and Hutchinson's teeth (affecting the permanent incisors), and these together with the signs present since infancy (hr agades, saddle shaped nose etc.) and Wassermann reaction will give the diagnosis.

Case report.—A male infant, aged about two months, was brought to a hospital on account of swelling and tenderness of the upper part of the right leg. He did not appear to be acutely ill. He was slightly febrile. Blood count repeated several times never showed more than 11,000 leucocytes per cubic m.m. Blood culture was sterile. Still the surgeon-in-charge was convinced that it was a case of coccogenic-osteomyelitis and insisted on operation. Blood serum gave a positive Wassermann reaction. X-ray also suggested congenital syphilis, though there was no other symptom and tibia was the only long bone involved. The condition improved with anti-syphilitic treatment though the child died later on of gastro-intestinal trouble.

Delayed closure of the fontanelle is another important sign of congenital syphilis.

Joints Congenital syphilitic arthritis:—In children and young adults congenital syphilis may cause an arthritis like that caused by tubercle. The knees are affected most often and the disease is often symmetrical. If there is much synovial exudation fluctuation is detectable; if there is much gummatous deposit in the sub-synovial tissues the synovial membrane feels thickened and irregular. There is no pain and very little impairment of movement. The history and examination for other signs of syphilis, specially nerve deafness or interstitial keratitis, must be thorough, and Wassermann reaction must be tested. In infants congenital

syphilis may cause osteochondritis in the sub-epiphyseal plate of cartilage and adjacent bone; the epiphysis becomes separated from the shaft so that there is mobility and dull grating as if fracture has occurred. At the same time there is considerable swelling of the soft parts around, from the inflammation having spread to them, so that there is much swelling about the joint although the joint itself is usually not implicated. Separation of the epiphyses from the shaft makes the limb unusable, hence the phrase syphilitic-pseudo-paralysis is applied to this condition. Suppuration is rare. There is rapid improvement with anti-syphilitic treatment. The condition may appear at any period from one month after birth till the age of two or three years, but it is seen most often when the child is two or three months old. It may be multiple. There is some tenderness and slight pain. Other signs of congenital syphilis are generally present, but if not, and the condition is suggestive, the child should be treated with Mercury and Penicillin and the result watched.

Gastro-intestinal system:—In some cases the mother has shown no obvious evidence of syphilis and yet is able to suckle her child without harm, even though there are ulcerating lesions on the child's gums and lips, whereas a healthy wet nurse develops a chancre of the nipple. This is called Colle's Law and was enunciated by him in 1837. Wassermann reaction in the mother has given a positive result in ninety per cent, it is therefore practically certain that the apparent maternal immunity is in reality due to previous mild and unrecognised infection.

The teeth in inherited syphilis are sometimes very characteristic. The temporary teeth usually appear early, are discoloured and crumble away. The permanent teeth are often sound and healthy but are sometimes deformed. The central incisors of the upper jaw are those most particularly affected, but the upper lateral and the incisors of the lower jaw may also be involved. Instead of being broader at the crown than at the roots they diminish in size from root to crown, being situated and separated from one another by interspaces. The angles of the crown are rounded off and a distinct notch forming the large segment of a small circle occupies the centre. The enamel is often imperfectly developed, hence they decay early. Occasionally they may be shaped like a screw driver narrowing from root to crown and a straight free edge. These notched and stunted teeth are sometimes known as "Hutchinson's teeth" but they are not now commonly seen.

Salivary glands:—Amongst the rare manifestations of congenital syphilis perhaps the most interesting are enlargement of, or other manifestations in connexion with, the salivary glands, particularly the parotid gland. Mostly it is the parotid gland that has been affected but others have also been involved. The ages at which this involvement has taken place has varied from two year

to fifty years. Mostly the attending surgeon, physician or the dental surgeon has not been sure whether the swelling was in the lymphoid gland, in the parotid or the parotid gland itself.

Case report.—A congenitally syphilitic mother aged about 25 years, when three months pregnant, got a swelling of the left parotid of the size of a small walnut. She consulted doctors who could not make out as to what was the nature of the swelling. Blood serum was never tested. The swelling gradually subsided in about three to four months. She had no anti-syphilitic treatment. After parturition she took the child to a hospital as congenital syphilis was suspected. She also mentioned about the swelling she had in her left parotid region. On examination she had shotty nodules in the left parotid region. Blood was sent for Wassermann reaction and reported to be strongly positive. Anti-syphilitic treatment was resorted to and the swelling cleared. Kemp and Moore have reported syphilitic parotitis and mastitis occurring in the same individual. More frequently, however, sub-maxillary and sub-lingual glands have been affected either alone or along with parotid. This may be brought about by an oral chancre. They are of gummatous nature.

Adam in 1939–'40 contributed an article to the *New Orleans Medical and Surgical Journal* under the title of "A Presumptive Sign of Syphilis" in which he says that in all the early and many of the late cases of syphilis he has noted changes in Stenson's duct, oedema or congestion or swelling of the duct and adjacent area and an erythematous inflammation of the orifice. The pathology he thinks is to be explained by the swelling of the lymph nodes which are present in the substance of the parotid and which presumably participate in general adenopathy of syphilis. This may in time interfere with the flow of saliva from the gland and so lead to inflammation about the orifice of Stenson's duct. Adam is of opinion that the ascending lymphatic or hæmatogenous infections of low grade character result as shown by the sign he describes.

Cardio-vascular system:—It is generally accepted that in acquired syphilis Treponema Pallidum is a potent factor in the ætiology of aortic aneurysm and aortitis as well as of heart disease and there is no *apriori* reason why the same should not apply to congenital syphilis. There is much divergence of opinion amongst clinicians, pathologists and radiologists on the subject and despite the fact that in 1943 Henrichsen was able to publish a review of 157 papers on cardio-vascular involvement in congenital syphilis, we seem to be no nearer to the solution of the problem today than we were in 1906, when, as Henrichsen tells us, five authors reported that they had found Treponema Pallidum in the heart of the congenitally syphilitic infants.

When an infant is born with manifestations of congenital syphilis it may be so ill that involvement of the heart may not be diagnosed clinically, and at autopsy, in the absence of any localised heart lesion, interstitial myocarditis may be overlooked.

Aortitis with or without aneurysmal dilatation has been described in syphilitic still-births and very young infants by several observers. X-ray examination of the heart and great vessels has been claimed by the Beretervides to be of great value in the diagnosis of syphilitic affection of these organs. An increase in the diameter of ascending aorta from the normal 1 to 1·3 cm. (according to age from 2–14 years) to 1·5 cm. or more being regarded by these observers as certain sign of syphilis. Although there have appeared several publications which appear to confirm the findings of the Beretervides, the matter must, for the present, be considered to be subjudice.

It is generally believed that in acquired syphilis the interval between the date of infection and the onset of symptomatic cardio-vascular disease is from 15–25 years. So, if the same is true of congenital syphilis the symptoms and signs of cardio-vascular involvement will not appear until the late childhood or adolescence or even later. Turnbull has reported two such cases, one in a girl aged 17 and the other in a girl of 7. Albut has recorded an instance of an abdominal aortitis in a congenitally syphilitic girl aged 16 years.

Whatever doubts some authorities may have as to the occurrence of congenital syphilitic aortitis, there can be no doubt about the frequent occurrence of specific arteritis in the peripheral blood vessels of these patients. The arteries at the base of the brain are those most commonly affected and since Sir Thomas Barlow in 1877 reported two cases in children aged 10 and 15 months respectively, there have been many similar cases reported in which serious cerebral symptoms such as hemiplegia, epileptiform seizures and mental deterioration preceded death.

Browning, Fromalavy and many other observers have reported positive Wassermann reaction in as many as 50–72 per cent of the patients and/or their mothers, whereas most American observers conclude that congenital syphilis is not an important factor in the causation of congenital heart disease. It is probable that some of the cases of cardio-vascular disease in young adults and even in the middle aged, ascribed to rheumatism or to acquired syphilis, may really be due to congenital syphilis—stigmata such as Hutchinson's teeth which may originally have been present having disappeared. Some patients, on the other hand, never exhibit any stigmata, yet a careful enquiry into the family history may elicit a specific background such as father's death from G. P. I. or from aneurysm or evidence of congenital syphilis such as interstitial keratitis in a brother or sister. The patient's serum reaction may still be positive

which will help in the diagnosis of syphilis (but not between congenital and acquired), or the serological reaction may have become negative with efflux of time.

Blood:—The Wassermann reaction may be negative in infant's blood at birth but may become positive some four or five weeks later. In this connection it is interesting to note that although the infective lesions may be present in the maternal passages, primary chancres are not seen in infants, who are presumably protected either by previous infection or by the presence of vernix caseosa. The Wassermann reaction in congenital syphilis is usually positive in the earlier more active stages. In some cases, however, in which the child appears healthy at birth the reaction may be negative but may become positive a month or more after birth. As the child grows and especially about puberty and in adolescence it may be diminished or be absent as in the later tertiary stages of acquired variety.

It should be realised that a large proportion of fathers of children with congenital syphilis have negative Wassermann reaction (Jeans and Cook in 1930 said it was 40 per cent of fathers whereas Nabarro in 1949 in a series of 123 fathers showed 60 per cent negative).

Eyes:—Interstitial keratitis or diffuse inflammation of the cornea occurs usually about the age of puberty or earlier. Diagnosis of syphilis in children is thought of by a steaminess of the cornea which usually appears soon after the child has celebrated his first birth day. It is limited at first to one eye, but the other is almost certain to be similarly affected at a later date. It commences as a diffuse haziness of the cornea which looks somewhat like a ground glass associated with the hyperæmia of the ciliary region. Red areas or 'Salmon patches' may be produced in the midst of the opacity due to a new formation of minute vessels. There is no tendency to ulceration, but in protracted cases the anterior part of the eye may project forwards constituting a condition known as "Anterior Staphyloma". The inflammation may spread to the iris and ciliary body. With suitable precautions the cases usually do well, although treatment for several years may be necessary and some corneal opacity may persist.

Interstitial keratitis associated with late congenital syphilis does not respond any better to Penicillin than to other previously tried remedies and final results leave much to be desired. In this condition Penicillin therapy should be supplemented by fever therapy routinely if possible.

Case report.—A Brahmin girl, belonging to a middle class family, aged about seven years, on joining a school, was noticed by her teacher to be unable to read the class black board from her seat. She was advised to consult a doctor. On examination both

the eyes showed haziness of the cornea. Iris were also involved. Her blood was therefore sent for Wassermann reaction. It was reported to be strongly positive. Anti-syphilitic treatment (Arsenic and Bismuth) was started. After about 18 months of treatment during which she had two attacks of malaria also she could read 6/24. This was in the pre-Penicillin era. Early in 1947 she was given a course of Penicillin injections. Unfortunately, soon afterwards, owing to disturbances in the Punjab, she had to leave her home and contact could not be maintained. Latest enquiry from her father reveals that she is attending her classes alright. No information about the acuity of vision is available at the moment.

Anti-histaminic substances have been found to be of some value in the treatment of interstitial keratitis.

Ears:—Infection of the middle ear *via* the Eustachean tube occurs as a result of rhinitis and may lead to the necrosis of the ossicles or that of the internal ear and may lead to deafness, specially about the age of puberty.

Syphilitic disease of the internal ear may produce symptoms closely resembling those of Menier's disease, in that giddiness, tinnitus and labyrinthine deafness are associated, onset being quite sudden. Vertigo is however occasionally absent. The trouble is usually unilateral and may occur in secondary or tertiary stage. The diagnosis depends upon the history of syphilis or other evidences of the disease. Similar symptoms may occur in congenital syphilis, usually between the ages of ten and fifteen years, though occasionally much later. Eustachean tube obstruction is present also, but the treatment of this fails to improve hearing. Other concomitant troubles, specially interstitial keratitis, may be found also.

The *spleen* of a small child is just palpable without there being any disease at all. If it is more decidedly enlarged the first suspicion will be that it is due to rickets or congenital syphilis. Congenital syphilis may be suggested by a knowledge of the family history, by the occurrence of snuffles, of specific skin eruptions; but in many cases the diagnosis will be one of surmise only unless confirmed by Wassermann reaction.

Central nervous system:—Cases of juvenile general paralysis have been recorded (Nabarro). Generally there is some damage to the brain. It may vary from a harmless ament to a ravenous lunatic, depending upon the stage at which diagnosis was made and treatment adopted.

Case report.—E, a girl of six years, was admitted in a children's hospital suffering from a mental disorder. At the time of admission the mental disorder was of a short duration—not more than three weeks. It was, however, reported by her teacher that the child was mentally peculiar since she first attended the school some

months before admission to the hospital. She was diagnosed to be a case of juvenile G.P.I. with characteristic findings in the blood and cerebro-spinal fluid. For eighteen months she was given energetic anti-syphilitic treatment. This was of course in the pre-Penicillin era, so the child got intravenous Neo-Arsphenamine, Bismuth, Mercury and malaria fever therapy. At the time she left the hospital the blood and the cerebro-spinal fluid were not quite normal. But since then she has been followed up and though alive has very limited mental capabilities.

PREVENTION.—Congenital syphilis can be prevented completely by ante-natal treatment which should consist of Penicillin and Bismuth but not Arsenicals. Special technique was needed for lesions such as interstitial keratitis.

The value of Penicillin in preventing the passage of syphilis from mother to child approaches perfection. Its ability to readily permeate the placenta makes it possible to adequately treat and cure the fœtus already infected in utero.

In pre-Penicillin days prevention of congenital syphilis was achieved admittedly with a certain amount, though small risk to the mother, by means of arsenicals and heavy metal treatment. The advent of Penicillin has enabled practitioners to administer a drug which is non-toxic and over much shorter period of time than was formerly possible. The dosage recommended by Goodwin and Moore is not less than two to four million units in aqueous or saline solution administered in divided doses given at intervals of three hours for $7^1/_2$ days. With the introduction of more modern preparations, however, one daily injection of Penicillin G, Procaine and Ammonium Monostearate in water or oil is acceptable and the woman need not be kept for more than ten days under the effect of Penicillin. The treatment should be followed by monthly clinical examination of the patient including a quantitative serological blood test. In the event of clinical or serological relapse, or if the original titre of the blood does not significantly decline within three months of treatment, the course is repeated. The authors found that the results were almost 100 per cent successful, and 42 out of 49 infants were followed up for six months or more which they consider long enough after birth to make practically certain of the diagnosis of "no syphilis". These results in the prevention of congenital syphilis are better, they say, then any obtained hitherto by any other method whatever. They state further that there is no satisfactory evidence that Penicillin is directly or indirectly responsible for abortion. Lastly, they recommend that in syphilitic expectant mothers Penicillin should be used as a routine to the exclusion of all other methods for treatment of congenital syphilis.

In the pre-Penicillin era we were taught that a syphilitic woman should receive adequate anti-syphilitic treatment in each pregnancy. This question *i.e.*, whether a syphilitic woman who has had adequate

treatment for syphilis need be treated in every subsequent pregnancy appears to be answered in the negative by Goodwin and Farber as a result of their study of 596 pregnancies in untreated mothers; 549 (92 per cent) of the children were born alive and of more than 70 per cent of these who were followed up for upwards of one year not one was found to be syphilitic. Of the 48 infants still-born or miscarried 20 were examined at necropsy and there was no sign of syphilis to be seen in any one of them. They conclude, therefore, that it is safe to withhold treatment of syphilitic expectant mother regardless of: (1) Stage and duration of her infection at the time of her original diagnosis and treatment; and (2) of the interval between the previous treatment and the pregnancy in which it is contemplated to omit the treatment provided that:

(*a*) That the mother has previously received four grammes of Arsphenamine (or its arsenical equivalent) together with concomitant Bismuth or two to four or more million units of Penicillin given for early syphilis in herself (this probably holds good also of late latent syphilis in the mother) and whether this treatment was given during the earlier pregnancy or during a non-pregnant interval; (*b*) the mother shows no clinical sign of syphilis; and (*c*) the mother's blood is negative, or, if positive, in low titre. Even a small amount of treatment with either Arsenic and Bismuth or Penicillin was able to produce a markedly favourable effect on the outcome of pregnancy in syphilitic women.

It is really too early to say that the procedure to give anti-syphilitic treatment to a syphilitic woman in each pregnancy should be cancelled in a woman who had Penicillin treatment. The short term results appear to be very good, but we must wait for 10, 15, 20 and even 25 years before the late results can be assessed, particularly the effects on cardio-vascular and nervous systems of the body.

The likelihood of a favourable outcome of pregnancy increases with the duration of syphilis irrespective of treatment.

TREATMENT.—The treatment of congenital syphilis should commence as soon as definite manifestations of the disease are present. When either of the parents is known to be syphilitic, the treatment should be carried in ante-natal life through the mother.

In the early years of this century treatment of congenital syphilis was by the time-honoured drug—Mercury, which no doubt seemed to act as a charm in many cases. The treatment lasting from a couple of weeks to a couple of months. It was, however, not realised at that time that this cure was not real but only apparent and that after a period of latency which may be three or four years, or might be ten or more years, the manifestations of late congenital syphilis will arise in the form of eye, ear, bone or mental disease. During the period 1914-'18 War Treponema Pallidum appears to have been re-invigorated to the introduction of new strains or by some

other evolutionary process, so the *severe* cases became more frequent and many died inspite of attempts at treatment with the new drugs —the Arsphenamines. Others, however, died not inspite of but because of treatment, or of a Herxheimer reation, though in all probability they would have died in any case from the intensity of their infection. Those who recovered after the combined treatment with Arsenic, Mercury and Bismuth appear to be really cured and some of them have been followed for 5, 10 and even more years during which their annual overhaul including a blood test ramained satisfactory. The ante-natal treatment of expectant mothers has to a certain extent masked the cases of congenital syphilis, unfortunately much to the detriment of such cases. This should not be taken to mean that I am against ante-natal treatment. I have been doing it all the thirty years of my practice and emphatically believe that it should be undertaken as soon as the pregnancy is diagnosed and that it should be carried out as efficiently and for as long as possible during the pregnancy. If that is not done infant's syphilis may not have been prevented but only rendered latent unless it is specifically looked for, and, if untreated, may manifest itself years afterwards.

So far as the treatment of congenital syphilis is concerned we all are agreed, I think, that the earlier it is undertaken the better the prognosis for the patient. Some auth rities maintain that if the treatment is delayed until after the patient is six months old we can never be certain that a cure will be effected. This may quite well be true, but in view of the advance which the treatment has made during the last few years this statement can be kept in abeyance. It is too early to accept it or to reject it. Syphilis is a difficult disease of which one cannot be dogmatic. According to Warthur, until one has made careful and extensive post-mortem investigation of the patient's organs and tissues, we can never declare that a patient has been cured of syphilis.

Every medical practitioner has treated with Arsenic and Bismuth a number of patients, some of them several years old when the treatment was started, who were apparently cured, who subsequently married and have healthy children. In cases where treatment was started prior to the fourth month of life clinical and serological results approached perfection (hundred per cent). In older children, however, the clinical response has been good but the reversion of serological test to negativity has been less satisfactory.

The older methods of treatment are known worldwide. I have to confine my remarks to the Penicillin treatment. In the United States of America treatment of congenital syphilis is carried out almost entirely, if not entirely, with Penicillin alone, with, according to published reports, uniformally successful results. In this country most authorities prefer to give combined treatment with Arsenic, Bismuth and Penicillin. American reports recommended

400,000 units once every 24 hours for 15 days. In cases with bone lesion higher doses have been given. The consensus of opinion is that most satisfactory results are obtained in young patients, those under three months' old. The rate in the fall in titre of the serological reaction seemed to bear no relation to the amount of Penicillin used, in the under three months group of patients. On the whole the results so far recorded are quite satisfactory, whereas cases of older children at about the age of five years with symptoms of neuro-syphilis and spinal fluid changes have not done well.

It must, however, be noted that the number of cases of congenital syphilis now encountered is much less than it was before the Second World War and the characteristic signs of congenital syphilis are rarer to encounter. Effective dosage of Penicillin given to 663 women with early syphilis during pregnancy resulted in 92·5% normal full term living infants and only 1·5% of living syphilitic infants.

The exact method of treatment with Penicillin is a matter of individual preference or expediency in meeting existing conditions, provided an adequate maternal fœtal tissue level is maintained for some seven to ten days. The most practically tested course for average use would consist in 600,000 Oxford units Procaine Penicillin G in oil with two per cent Aluminium Monostearate once daily for 10 days. Less frequent injections, for example, one to two million Oxford units every other day to a total of six million Oxford units, has yielded similar results with a smaller series of cases.

An analysis of 267 pregnant women treated for early syphilis with more than ten weeks' Arsenicals with or without Bismuth in the period immediately preceding the introduction of Penicillin yielded results not statistically different from the Penicillin-treated group. Nevertheless ease of administration and short duration of therapy, the lack of toxicity and the ability to cure in utero the already infected fœtus, makes Penicillin alone the preferred treatment in the prevention of congenital syphilis.

The mortality rate was 17·5% in Penicillin-treated syphilitic infants less than four months of age. These deaths all occurred during the course of Penicillin therapy or shortly afterwards. It was felt that the death was the result of debility rather than of the treatment. The general health must therefore be attended to, and if the mother is unable to nurse the child it must be put on the bottle. On no account must it be given to a wet nurse. So, supportive pædiatric care is of extreme importance in debilitated infants.

Many syphilitic infants during the first year of life, die from malnutrition or marasmus, but if properly treated a considerable proportion regain their health, within six or eight months, all the manifestations mentioned above disappearing, although their scars

remain. The child's subsequent development is impaired and it often retains almost pathognomonic facies.

Some abuses in the treatment of syphilis were:—

(1) Overdosage—specially in cases of congenital syphilis and latent syphilis with fixed positive serum reaction.

(2) Under treatment of early syphilis often without established diagnosis.

(3) Local application of Penicillin.

(4) Treatment of healthy persons wrongly suspected of having syphilis.

Penicillin has established its place in the treatment of syphilis and with few exceptions should replace Arsphenamines. Bismuth compound should be continued with it in all stages. In late syphilis, treatment with Bismuth should precede Penicillin to reduce the risk of Herxheimer reaction and therapeutic paradox.

Special features.—The length of time a syphilitic patient of either sex retains the power of transmitting the disease to the fœtus is an exceedingly difficult point to determine and one which is constantly coming before a practitioner, who is asked to decide in what period marriage is safe. The rule of practice generally followed is that no one suffering from syphilis should be allowed to marry until Wassermann reaction has remained negative and he or she has been free from all symptoms for two years.

In a few cases, however, a positive reaction may persist inspite of up-to-date treatment. In such cases, in the absence of all clinical signs, it is probable that the risk of marital infection would be slight. It will be wise, however, in the case of a subsequent pregnancy to put the mother under treatment.

The question of transmission to the third generation has been one of much interest concerning which a good deal of conflicting evidence has been forthcoming. The dependence of this disease upon a recognised organism which it has been possible to demonstrate in late tertiary stages, is presumptive evidence in favour of its transmissibility, but naturally one of the chief difficulties in the proof of absence of re-infection in the second generation.

Whereas we are making great strides in the reduction of cases of congenital syphilis, we should now aim at the virtual extinction by adequate ante-natal investigation and treatment of the expectant mother. My belief is that by so doing we should not only reduce congenital syphilis to a minimum, but might also reduce the incidence of congenital deformities and malformations in addition to obscure diseases of infancy and childhood which possibly result from untreated or undertreated maternal syphilis.

We have in Penicillin a remarkable and potent remedy for the treatment of syphilis. Although the short term results are very felicitous, we must not lose sight of the fact that it will be many

years before we can express a confident opinion as to the claim that Penicillin is the best for syphilis. In this country one of the most difficult obstacles to an adequate enquiry into the subject is the difficulty of maintaining contact with patients over sufficient number of years, practically the remainder of the patient's life.

It is suggested that the Penicillin-treated patients to be followed up should include the following categories :—

1. Patients with primary or secondary acquired syphilis.
2. Patients with acquired cardio-vascular and neuro-syphilis.
3. Congenitally syphilitic infants who started treatment under six months.
4. Congenitally syphilitic infants who started treatment over six months.
5. Children with congenital neuro-syphilis.
6. The clinical and serological follow-up for as long as possible of children born after the mothers who had ante-natal treatment with Penicillin as stated above.

"Congenital syphilis is rapidly ceasing to be a serious medical and public health problem where good control measures are possible. Still it will continue to be a threat as long as syphilis is prevalent in any degree in the adult population. With modern public health practice and the effectiveness of Penicillin during pregnancy, the crippling death-dealing impact of infantile and late congenital infection results almost completely from neglect in the application of our knowledge. The fact that an appreciable number of infants or children with prenatally acquired infection exists shows that even in areas where general public health practice is considered to be good too many cases of syphilis are escaping detection in marriage and pregnancy. While this is not always the fault of the physician who handles the individual case and may often be attributed directly to the shortcoming of the patient, yet, it does represent a defect in social organisation which can be remedied to the benefit of civilization and all mankind" (Ingraham).

Congenital Syphilis in Twins

Nonidentical twins with congenital syphilis were affected in different degrees. The boy had a syphilitic coryza at the age of 4 months; at the age of 6 months he died with pneumonia in spite of penicillin therapy. Besides the coryza, a periostitis of all long bones and a gumma of the right upper mandible were found. At postmortem examination spirochætes could not be found. The other twin, a 7 month old girl, had no clinical signs of syphilis. A slight periosteal thickening of the diaphyses of the humeri was noted, and the Wassermann reactions of the cerebrospinal fluid were negative, but the "luetest" elicited a positive reaction. The infant was treated, and on re-examination after six months, only the "luetest" reaction remained positive. The mother, a primipara, had no clinical signs of syphilis and was never treated. The blood serum and the cerebrospinal fluid Wassermann reactions were positive. The father's blood serum Wassermann reaction was repeatedly negative The literature is discussed. Perhaps the most interesting feature is that in cases of nonidentical as well as of identical twins it has been reported that one of the twins did not have the disease —H. Lauseeker, *Wien. klin. Wchnschr.* 63 :85 (Feb. 3) 1950.—*American Journal of Diseases of Children.*

OPHTHALMIC PROBLEMS IN INFANTS AND CHILDREN

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MULTIPLE ophthalmic problems come up for consideration during clinical examination of infants and children. Most infants are born with a pair of good eyes—the eyes of tomorrow. It is the responsibility of the parents, the family doctor, the specialist and the State to see that these eyes last him for his life time.

Firstly, before birth one has to look after the mother. Her nourishment, general state of health, anæmia, clothing and housing should receive special consideration. Prenatal examination is important and this should always exclude blood against syphilis which takes quite a large toll of children's eyes.

As soon as the child is born we have to safeguard against ophthalmia neonatorum. Preventive measures in this condition have yielded good results. Blindness from ophthalmia neonatorum has been reduced to a negligible minimum in most of the countries. Though Crede's system is still practised far and wide over the globe, attempts at combination of Penicillin therapy with Crede's system has yielded much better results. When the disease is established Penicillin 1,000 units to a c.c. given as drops every five minutes for the first two hours and later every hour has yielded most amazing results, in that the child is able to open the eyes in 12 to 24 hours and the organisms almost disappear as seen in the smears after 24 hours.

Some premature children present a condition called 'Retrolental Fibroplasia'. The term was first used by Terry in 1943, but later some confusion was made and other conditions like 'persistent hyperplastic vitreus' and 'retinal dysplasia' were mixed with it. Retrolental fibroplasia affects children of premature birth and those born with low birth weight. The disease occurs in two stages: (*a*) the acute stage occurring between the third and fifth week of life comprising of dilatation of retinal veins, hæmorrhages, transudation and formation of new vessels in the vitreus; and (*b*) the cicatricial stage in which the eye is contracted and shrunk after organisation. It is a manifestation of congenital angioplastic process of premature infants and it possibly may be related to a deficiency of the adrenal steroids. The condition is bilateral and the therapy is still a matter of conjecture. However, all premature children, and children with low birth weight, must be kept under observation during the first two years of life.

In a majority of cases a congenital birth cataract has been observed and this has been attributed to, and satistically proved to be due to, the mother developing a virus infection, mainly Rubella,

between the 8th and 12th week of pregnancy. This is the time when the sense organs and the viscera are being differentiated.

During the first two years the eyes have to be guarded against infections like conjunctivitis, ulcers cornea, and also against avitaminosis. Every child with an infected eye must be given a thorough examination, if need be even under an anæsthetic, to ascertain the nature of the disease and the condition of the cornea. The present day advance in therapy has done away with that drastic touch of Silver Nitrate which makes the child shun the doctor. Today Sulphacetamide soluble, 10%, Penicillin 2,000 units per c.c. and ocular Aureomycin and Terramycin for local instillation are the therapies of choice, and yield much better results. The ulcer must be cauterised with either Acid Carbolic, or absolute Alcohol or Tincture Iodine 7½% and Unguentum ½% instilled locally. (Atropine as a solution should be avoided in children).

Amongst disorders of malnutrition are the deficiency of vitamin A which leads to xerosis of conjunctiva, keratomalacia, lack of tears, and deficient dark adaptation. Vitamin A deficiency must be considered as an ophthalmic emergency for purposes of treatment. Large doses of vitamin A must be given parenterally (100,000 to 300,000 units) and Cod liver oil in the eyes and by mouth. Riboflavin deficiency leads to a chronic kerato-conjunctivitis with superficial vascularisation of the cornea. (*Fig. 1.*) Replacement of the vitamin is the obvious treatment. Deficiency of vitamin C causes hæmorrhages in the eye and eyelids. The eye may show a bruised appearance. Lack of vitamin D may result in a zonular cataract.

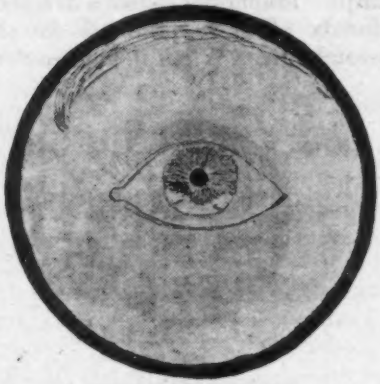

FIG. 1.
Keratitis from Ariboflavin deficiency.

In considering these it must be realised that little care and simple preventive measures can lead to great improvement in the ocular wealth of the nation.

The age of kindergarten school is a special time to watch. Here teachers at the school can be most helpful. The child cannot read, but simple training can make them point to the direction of E and C and an expert teacher can always guide the parents about the health of the eyes of their children. Cross eyes should be treated promptly. If the child has cross eyes it should not be neglected, it is ruinous to the future of the child. For, when the child realises that his eyes are

not like those of others he feels shy, other children tease and torment him. His health suffers and he develops an inferiority complex. Most of the cross eyes between the age of 2 and 4 years can be corrected and straightened and a normal binocular vision can be developed. After the age of 4 years it is difficult and generally results in individuals possessing a blind cross eye.

At the same age one common condition met with is phlyctenular keratitis and conjunctivitis. Most of the children suffering from phlyctenules show a certain amount of associated keratitis. The cause of phlycten is still unknown. Fritz ascribes it to staphylococcal infection as a common cause and tubercular allergy as a second cause. Experimentally one can produce a lesion resembling phlycten by sensitising conjunctiva to a wide range of bacteria and proteins. Histopathologically phlycten is not a tubercular lesion. However, in diagnosing phlycten a tubercular lesion in another part of the body should never be lost sight of.

Diseases like trachoma and spring catarrh may present difficulty in early diagnosis. Every child who is a contact of trachoma must be thoroughly examined with slit lamp. Trachoma follicles are seen in the early stages in the upper fornix (*Fig. 2*). Smears in the early stages may show the cell inclusion bodies. When it has reached

FIG. 2.
Early follicles in upper fornix.

FIG. 3.
Membrane on the cornea in a case of trachoma.

the 4th stage a diagnosis is too late. Another rare complication is a dense membrane on the cornea in some cases of trachoma (*Fig. 3*). In the early stages treatment should be commenced with Sulphacetamide 10% locally and preparations of Sulpha orally, the dose depending on the age of the patient. Ophthalmic solutions of Terramycin and Aureomycin are also useful. Spring catarrh is more a manifestation of allergy and should be treated on those lines.

Preventable and communicable diseases.—Smallpox can be prevented by primary and regular secondary vaccination. Should the disease crop up, always safeguard the cornea as smallpox is a common cause of thick corneal opacities following ulceration of the cornea. Diphtheria is another condition which can be prevented. If the child does get diphtheria he may develop no complications of

the eye at all or he may develop conjunctivitis, paresis of accommodation, or paralysis of one or more of the extra ocular muscles. Subconjunctival hæmorrhage occurs in whooping cough and clears up without any specific treatment.

Should syphilis be acquired congenitally the child may develop interstitial keratitis at the time of the second dentition, though the condition has been observed in earlier years as well. It is important to safeguard the child before birth by an examination of the mother's blood during pregnancy and, if necessary, her treatment; similarly a vaginal smear can help to exclude a gonococcal infection and its after results on the child. Prevention is always better than cure. When interstitial keratitis does develop, attention should be paid to other signs, like Hutchinson's teeth, frontal bosses, bilateral painful synovitis, nerve deafness and rhagades. The therapy of choice is Penicillin intramuscularly 10 mega units at the rate of 50,000 units four hourly or Procaine Penicillin 400,000 units twice a day. Atropine 1% should be instilled locally in the eye until the eye is quiet and ciliary injection and redness have disappeared. The Penicillin therapy should be supplemented with metallotherapy. In certain patients one may get uniocular keratitis. This is generally tubercular in origin, probably an allergic manifestation of it.

Visual incapacitation.—Every child must be tested for his or her vision in the pre-school age. Spects if necessary should be prescribed. Myopia should be fully corrected, and a myopic child should never be taken off the school. It is a gross injustice as it makes the child more introspective. The school authorities and the administration should consider it their duty to get the eyes of the children examined regularly. Poor vision in a child limits his experience. He becomes backward as he is unable to complete his task and as a necessary corollary one may say that he tries to isolate himself. A clever teacher should try to find out such defects and not call the child stupid. The remedy does not comprise merely in prescribing spectacles. Spectacles may help a majority of them to get a clear and good vision. There are several other considerations which should not be ignored by the school authorities, the administration, the School Boards and the Universities. Firstly, there is need for co-operation of architects in the construction of the modern schools. Old, dark and dingy buildings are ill adapted for the use as schools for children. Schools should be well planned and well constructed, due consideration being given to ventilation, light from the sky, illumination, the location and the size of the windows and the seating accommodation for the children. The black board should be away from the glare and no child should be allowed to sit facing a window. In certain weather, specially in monsoon and winter, the school room requires additional artificial illumination from the interior as the daylight is not adequate. Such lights should always be arranged in consultation with the Illumination Engineers. Other

important points for the poor vision child are the text books which should have a large print which is easy to make out, the pencils should have thick lead, and the boards should have special arrangements whereby it is not difficult for the child to see. Some beginning has been made towards the care of the visually incapacitated children but more effort is needed on sound lines to probe into the greatest needs and the greatest possibilities of improvement.

Injuries can occur at birth due to forceps delivery. This may damage the cornea, lead to paresis of extraocular muscles, or hæmorrhage inside the globe from compression. Between the age of 2 to 5 years children are fond of objects with a glitter. Sharp instruments with a glitter can cause penetrating eye injuries. In school age most of the injuries are due to games organised and unorganised. In our country a common cause of injury to the eye in children is the 'cat and the tail'.

Blindness is a catastrophe of major importance. The child is shut off from the world for the rest of his life. Blindness due to disease of the cornea may be treated in some cases by corneal grafting. The need of the day is an Eye Bank for the country. While there is much in the air about the prevention of blindness we need a concentrated effort on the part of the profession, the public and the State to organise a 'National Society for the Relief and Prevention of Blindness', to devote their energy to the causes of blindness and its prevention in children. A blind child differs from the deaf child. Whilst a deaf child has a tendency to turn into a recluse, the blind is highly intelligent and would like to utilise his prowess in a way that would make him a useful member of the society. Rehabilitation claims to answer our query. Our experience in the last war has shown that the blind can be trained in several useful arts and crafts and though handicapped for sight it is surprising how much they can do and in some cases even better than normal individuals. Thus they can be turned into citizens useful both to themselves and to the nation.

It would appear from the foregoing pages that most of the ophthalmic problems in infants and children are vital problems of health. It is the duty of every one to safeguard the valuable pair of eyes and thus lay an edifice for the future; as Lord Beaconsfield remarks, "Public Health is the foundation upon which is built the happiness of the people and the power of the State".

XEROPHTHALMIA AND ALLIED CONDITIONS

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"XEROPHTHALMIA is the principal cause of blindness in India", wrote Wright and Kirwan in the year 1931. In those days we neither had the antibiotics like Penicillin and Aureomycin nor chemotherapeutic drugs like that of the Sulpha group, to fight other infections causing blindness. We had no food shortage, and nobody had heard of ration cards. Family planning and contraception were topics confined to the fashionable drawing rooms of the sophisticated few. If the above quoted statement was true in those days (as it undoubtedly must have been, coming from such authoritative sources), how much more true it must be today ? This is an enormous problem which must be tackled both by our politicians and members of the medical profession.

Xerophthalmia is essentially a disease of children though a few adults are also affected. Xerophthalmia and allied conditions, night blindness and keratomalacia are due to deficiency of vitamin A in the body. There are other factors which may cause the above symptoms; and these are of such rare occurrence that they will not be discussed in this article.

Vitamin A deficiency may be caused by :—(*a*) Insufficiency in diet; (*b*) improper absorption; and (*c*) increased demand by the body.

The first factor is of such common occurrence in our country that in the vast majority of cases it may be put as the main cause. Poverty and ignorance of proper dietetic habits are mainly responsible for this.

Many intestinal diseases, liver diseases etc., cause insufficient amounts of vitamin A to be absorbed and thus result in deficiency symptoms. In a tropical country like ours, diarrhœas, dysenteries etc., are so common that this is also one of the major causal factors.

During active growth period, and acute illnesses like infectious fevers, demand by the body for vitamin A is increased. Many of us still have the pernicious habit of starving our fever cases, that the danger of causing deficiency complications are very real. If smallpox or measles attack a child, in the majority of cases a doctor is not even called in. In my opinion many cases of blindness after smallpox are due to keratomalacia as a result of semi-starvation in the face of an increased demand for vitamin A.

Some of the other abnormalities caused by vitamin A deficiency are :—(1) Changes in skin ; (2) changes in other mucous membranes and nervous system ; and (3) reduced resistance to infection, etc.

But the commonest and earliest symptoms in children are of the eyes. These only will be considered here.

Delayed dark adaptation is the first eye symptom caused by vitamin A deficiency. Detection of this needs complicated apparatus in expert hands. In ordinary clinical practice we never come across such early cases.

(1) Xerosis with or without typical Bitot's spots; (2) night blindness; and (3) keratomalacia are the types of cases we usually come across.

Xerosis.—In this condition the lustre of the conjunctiva is lost and it is thrown into folds. The tears fail to wet the affected parts of the conjunctiva. Frequently the colour of the eyes is dirty brown. In mild cases only patches of conjunctiva, especially in the area of the palpebral aperture, may show dryness. These children need not be conspicuously ill nourished. *Bitot's spots* are whitish foamy triangular patches seen in the area of palpebral apertures especially on the lateral sides. These are also not wetted by tears.

Night blindness.—This is usually associated with the above two symptoms, especially in children.

This may be of a mild degree resulting in stumbling of the child over obstacles at night or severe, many times described by the anxious mother that "the child gropes in his food plate at night." All the 12 adults with mild eye symptoms indicated in table A came to me with Bitot's spots. Only a few of them admitted reduced acuity of vision at nights. Evidently adults go in for treatment before gross night blindness sets in.

Keratomalacia is seen most commonly in infants. They are ill nourished and often marasmic. The conjunctiva is dry and Bitot's spots may be seen. The cornea may show various degrees of involvement from just dryness and a few whitish discrete infiltrations to gross melting corneæ with yellowish white slough. The cornea may be perforated with iris prolapse. As a contrast to this dramatic picture is the absence of other inflammatory signs. The lids are normal. There is that absence of marked redness and œdema that we often see with conjunctivitis and infective corneal ulcer. The important diagnostic sign is the ease with which examination can be made of the child's eyes due to absence of blepherospasm or œdema of the lids. This quiet appearance of the eyes is the dangerous feature of the disease, and is responsible for many mothers not consulting the doctor in time. "The child just kept her eyes closed for two days, doctor", wails the mother when we inform her that the eyes are irreparably damaged. The lesion, when once it sets in, develops with such rapidity that the whole cornea may slough away in one night. Associated with corneal ulcers may be hypopion.

Pathologically the above results are said to be due to changes in the epithelium of the conjunctiva and cornea which becomes epidermoid in character. The relentless rapidity with which corneal

damage spreads with absence of inflammatory signs lead many to believe that the changes are essentially neurotropic.

PROGNOSIS.—The milder cases show a dramatic cure with exhibition of vitamin A. I have seen night blindness disappearing within a couple of days of starting treatment. But occasionally Bitot's spots may persist for some time after the treatment is instituted, as a symptomless condition.

It is in the severe cases that one some time has to look on helplessly as the cornea is sloughing away and ugly staphylomatas form in spite of adequate treatment. The only redeeming feature is that when the two eyes are unequally affected, the less severely affected eye can be saved in the majority of instances. In some cases the patient is discharged blind with dense corneal scars occluding the pupillary areas. In these an optical iridectomy can be tried later, and, where facilities exist, corneal grafting. Of course the prognosis for vision is tied up with the general condition of the patient. It is indeed a blessing in disguise that many of the marasmic infants with hopeless prognosis for vision, soon die of their general condition.

One of the hopelessly blind cases—2 years after a severe attack of keratomalacia.

TREATMENT.—The most important thing to watch for is, whether the cornea is involved or not. If it is, the condition is an emergency and very active treatment must be instituted.

The following line of treatment must be followed :—

1. Local treatment of the eye condition.
2. General symptomatic treatment *viz.*, supplying vitamin A.
3. Treatment of the ætiological factors causing the deficiency.

Local treatment:—Local treatment is really very simple. If the condition is one of mild xerotic patches or Bitot's spots, they need no local treatment. If they are moderate or severe, a drop of Liquid Paraffin instilled twice a day keeps the conjunctival sac lubricated.

If cornea shows mild involvement by dryness and dull appearance the same treatment would suffice. If there is definite corneal ulceration, Atropine ointment 1% twice a day must be instilled. In these cases there may or may not be a hypopion, but since there is almost always a secondary infection I supply Penicillin eye drops (10,000 units per cc.) to be instilled in the eyes at intervals of every half an hour. I have not seen any special advantage in the local instillation of Cod Liver Oil. It can be used instead of Liquid Paraffin as a mere lubricant.

General symptomatic treatment:—If the cornea is not involved supply of vitamin A by mouth would do. Cod liver oil, and shark liver oil as such, or incorporated with malt as in many of the elegant patent preparations, can be given to older children and adults. In the younger age groups concentrated vitamin A and D liquids put up by many firms (Adexolin Liquid, Glaxo, etc.) can be conveniently given mixed with mother's milk or cow's milk. We have also capsules, pearls and other water miscible tasty preparations (Homicebrin, Lilly's), available to suit individual pockets.

If cornea is involved vitamin A must be injected at once. For this the following doses are available; 2500) units ampoules (A Vita. injectable, Philips), 40,000 units ampoules (A vitamin, C.D.C) 75000 units (Radiostoleum, B. D. H.), 100000 units ampoules (Prepalin, Glaxo) and 300000 units ampoules (Aravit, Roche).

If the cornea just shows slight dryness 25,000 units injected once a day for 3 days followed by vitamin A by mouth is sufficient. I have seen cornea brighten up in most of the cases within 24 hours after the first injection. If there is corneal ulceration I use 100,000 units injections once a day for 3–6 days, according to the severity of the condition and local reaction, if any, at the injection site. In some infants this local reaction can be very frightening indeed. Vitamin A by mouth can be given simultaneously or following the course of the injections.

In some cases concentrated vitamin A preparations by mouth, cause diarrhœa. Here again a couple of injections may be necessary to tide over the crisis.

In infants, rubbing of cod liver oil over the body is a useful measure.

As a part of general symptomatic treatment adequate quantities of vitamin C are said to be useful adjuvants in producing quicker healing of ulcers and consolidation of corneal scars. I always prescribe one tablet (100 mg.) of Vitamin C, crushed and mixed with milk, three times a day, in all my cases of keratomalacia. This is admittedly a smaller dose than what is advised (1000–4000 mg. per day).

It goes without saying that other co-existing deficiencies, if any, must also be adequately treated.

Aetiological treatment:—This is the real problem for the general practitioner. General diseases like cirrhosis of liver, enteritis, infectious fevers etc. must be simultaneously treated. Many of the infants with severe keratomalacia are in an extreme marasmic state and probably never survive for long. Correct dietetic habits must be taught to ignorant parents. It is a fact that many lay magazines and advertisers publish erroneous information about vitamins which are taken as gospel truth by the educated public and even by some members of our profession. In giving advice to our patients the following facts based on scientific research may be useful:

1. Vitamin A and Carotene are very far from being one and the same.
2. In health, and most certainly in disease, only a very small percentage of Carotene is converted into viatamin A in the body.
3. No vegetable contains vitamin A. Some of them like carrots, tomatoes and green leafy vegetables contain a lot of Carotene only. Even here, contrary to the popular idea, green leafy vegetables are a better source of Carotene than carrots because Carotene is better utilised by the body from the former source.
4. Vitamin A itself is found only in very few foods of which the commonest are butter, eggs, milk, liver and some fish.
5. In fevers there is no use of giving vitamin A by mouth. It must be injected.

PROPHYLAXIS.—This is a national problem tied up with factors like standard of living, education etc. In England, children and pregnant mothers are supplied with Cod Liver Oil, free of cost. The members of the medical profession and health authorities, wherever possible, should educate the public about correct dietetic habits. Pregnant women and growing children, must be supplied with extra vitamin A, as such. Lot of experimental research is being carried out now-a-days about fœtal abnormalities and many of the workers are of opinion that they may be due to the low level of vitamin A in the pregnant mother. German measles during pregnancy has resulted in many offsprings being born with congenital cataracts or other abnormalities. It is surmised by some that this may be due to deficiency of vitamin A during the period of illness. It is hence suggested that extra vitamin A should be supplied in pregnancy, especially during fevers of any type, when they must be injected.

LACTATION.—Here again ignorance is the cause of many debilitated mothers breast-feeding their children even when the latter are as old as three years. Lactating mothers should be given extra vitamin A and breast-feeding should not be continued in any case over a year. Supplementary nutritious feeding must be instituted in infants, if the mothers are debilitated, or are acutely ill.

In the following lines I am presenting a few facts about a small series of cases that I had come across in my clinic. As statistics

they are useless, in that the number treated is small, not representative in character and without adequate follow-up. But they give an useful indication about the type of cases met with in private practice.

In a series of 2,000 patients seen at my clinic, 268 were children under 10 years of age. Of these children, 30 cases were suffering from xerophthalmia of varying degrees *i.e.* a little over 11%. On the other hand, only 14 cases were met with among 1,732 older patients *i.e.* about 0·8%. I am sure that in a public hospital the percentage of such cases would be far greater, considering the class of patients that form the majority there.

TABLE A

Type of Cases	Out of 268 children			Out of 1732 older patients
	Below 2 yrs.	2–5 yrs.	6–10 yrs.	
Xerosis, Bitot's spots with or without night blindness.	...	3	3	12
Xerosis with mild corneal involvement.	1	3	...	...
Xerosis with severe corneal involvement.	15	3	2	2

It is seen from the above Table that infants below 2 years were always seen with corneal involvement.

TABLE B
Aetiology

Age group	Breast-feeding by debilitated mothers	Ignorance and bad diet	Cirrhosis of liver	T. B. enteritis	Infectious fevers	Other factors known and unknown
Below 2 years ...	5	...	4	...	2	5
2–5 years ...	1	4	2	...	2	...
6–10 years ...	...	1	...	...	2	2
Above 10 years of age	..	12	1	1	...	...

One tragic case was that of an infant 3 months old who had harelip and complete cleft palate with difficulty of feeding. The baby was just skin and bones and brought in with both eyes seriously involved, with perforated cornea and iris prolapse. The surgeon who saw the baby at birth is said to have advised operation six months later. This was unfortunate. The baby must have been operated soon after birth, however great the surgical risks might have been, if the baby could not be fed adequately by any other

means. To the great relief of the parents, the infant died within a fortnight of its becoming blind.

TABLE C

Results in severe cases after treatment

Result	Children			Others
	Below 2 yrs.	2–5 yrs.	6–10 yrs.	
Hopelessly blind ...	6	2	1	...
One eye saved ...	4	1	...	...
Hope of getting some vision by optical iridectomy.	3	...	1	2
Hope of vision after corneal grafting ...	2	...	...	...

More than half the infants below 2 years among the above cases were in an extremely marasmic state and probably never survived for long. Both the two adult cases shown above died within 2–3 months after seeing me, of their general diseases.

Conclusion and summary.—Xerophthalmia is a very common eye disease among children and is undoubtedly one of the major causes of blindness in India.

Lack of marked inflammatory signs in the face of extensive damage, is a characteristic feature of this disease.

Among the ætiological factors, improper diets, and acute infectious fevers are notable.

Very vigorous treatment must be carried out as an extreme emergency measure, when cornea shows any involvement.

Prophylactic measures such as provision of adequate vitamin A during febrile periods, pregnancy and lactation; and education of the public as to the correct dietetic habits, are stressed.

A small series of 44 cases of xerophthalmia is presented, their treatment and results are evaluated.

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OPHTHALMIA NEONATORUM

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THE purulent conjunctivitis of new-born is known as ophthalmia neonatorum. It is the most serious form of conjunctivitis occurring in the villages of India.

Once I had gone to see the Victoria Blind School at Bombay and, to my surprise, I found that more than 50% of the inmates had lost their eyes from corneal opacities due to purulent conjunctivitis in childhood. In my 25 years' experience in Surat, I find that lately very few cases are coming up from villages, possibly due to more trained nurses conducting labour cases in villages.

It is a preventable disease and since the advent of Penicillin and Sulphonamides, it is almost wholly curable.

SYMPTOMS.—Any discharge from baby's eyes during the first week should be viewed with suspicion, as tears are not secreted at this early date. In case of injection the discharge soon becomes muco-purulent and then purulent. Pus is welling into the lids. This is usually noticed on the 3rd day. If delayed beyond a week it is probably not a true case of gonorrhœa-ophthalmia. Conjunctiva becomes inflamed, there is marked chemosis.

If the disease is not brought into check at once, cornea is soon involved and there is rapid destruction of cornea.

BACTERIOLOGY.—The most common organism is gonococcus, but pneumococcus, influenza bacillus, staphylococcus, and the virus of inclusion conjunctivitis play their role.

PREDISPOSING FACTORS.—At birth eye has less resistance to injection than in later life and as lacrimal secretion does not appear until after few weeks of life, the infantile eye is deprived of the important defensive mechanism of tears (the powerful anti-bacterial enzyme-lysozyme).

CAUSATIVE FACTORS.—During labour the infected vaginal secretion contaminates the eye-lids and the dirty hands of nurse or native midwife add to the infection.

PROHPYLAXIS.—It is a notifiable disease in England and, I think, if the same law is applied in India, we would be able to save many children from the curse of blindness.

Prevention of ophthalmia is no new consideration. The essentials were laid down by Burjani-Gibson in 1807. The tragedy lies not in ignorance but in non-observance. The first essential is antenatal care to see that the vagina is not infected with some disease.

The next most important step is that a few drops of Silver Nitrate 5 grs. to an ounce of diluted water, must be instilled in the eyes of all new-born babies.

Here I may mention that Silver Nitrate is a far better curative drug than the new fashioned organic salts of silver (put by various companies in the market) in all forms of conjunctivitis.

TREATMENT.—There are few serious conditions in which Sulphonamides and Penicillin have achieved more dramatic results.

Whereas Sulphonamides has shortened the treatment of this condition from weeks to days, Penicillin has reduced it to hours. (Sorsby).

Where Penicillin is available, give Penicillin injections as in adults, except that the dose is to be reduced 5000 units every 3 hours (intramuscularly) till discharge stops.

The local treatment is instillation of Penicillin drops (2500 units 1 c.c.) every 10 minutes, until there is no discharge.

Irrigation of eyes with Normal Saline is not needed in this treatment.

Generally 1 hour to 3 hours treatment is quite sufficient before the eye is dry.

Swelling of the lids persists after cessation of discharge. Penicillin drops are now instilled every hour for 12 hours and then every 3 hours for the next 24 hours.

This Penicillin treatment is supplemented by any Sulphonamides (preferably Sulphadiazine) orally, ·1 grm. per kilogram body weight every 3 hours for the 1st day and every 6 hours for the 2nd day.

Where Penicillin is not available as in villages, Sulphonamide treatment orally as well as 5 p.c. ointment applied every hour to the eye, after irrigation with Normal Saline, is equally satisfactory.

Supplement this treatment by putting Silver Nitrate 5 grs. to an ounce solution once only every morning and putting Mercurochrome 1% drops in the interval every 3 hours. Of course if the cornea is involved one must put Atropine Sulphate 1% drops or ointment thrice a day.

Treatment of Tetanus in Childhood

In 28 cases of tetanus, five of which were in new-born infants, the treatment consisted in intensive serum therapy, with serum given fractionally; generous doses of phenobarbital derivatives, special care of the patients, and all possible forms of symptomatic and general therapy. Of the 28 patients, 21 were cured (75 per cent of the total); of the five new-born infants, three were cured and two died. With the systematic employment of the customary antitetanus therapy, the prognosis of this disease, which was once so serious in the field of pædiatrics, tends toward notable improvement.—Severino Pedone, *Riv. Clin. Pediat.*, 46; 833 (Dec.) 1948.—*American Journal of Diseases of Children.*

CONVULSIONS IN CHILDREN

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ONE of the simple but alarming conditions in children that a general practitioner is often confronted with is convulsions. Children up to the age of five or seven are very much prone to go into convulsions on the least upset in their health. In many cases the condition is comparatively harmless and easily treated. But to the general practitioner who is often called upon to treat these cases at home and at odd hours they are a source of annoyance and anxiety. Home treatment of these cases is very inconvenient except in very mild and simple cases.

The causes of convulsions in children are many. Diseases in which convulsions are a characteristic symptom, convulsions due to injuries to the brain and cranium during birth and idiopathic convulsions are not within the scope of this discussion. It is common experience that children get into convulsions in conditions which do not produce convulsions in the adult. This symptom in children in these cases is more or less due to, and characteristic of, their tender age and development.

In a series of 17 cases attended to in our Nursing Home during the last about eighteen months, one was a case of meningitis, one was of diphtheria and the rest were just fever with convulsions. Meningitis and diphtheria were diagnosed only after the child was admitted into the Nursing Home. It is always essential that a case of convulsions should be examined thoroughly in the routine and the real disease and the cause of convulsions established.

That the nervous system is not fully developed in children and so is comparatively less balanced and more easily upset is common knowledge. This naturally gives rise to convulsions in children for comparatively trivial irritations to the nervous system which in an adult would not bring about the same result. Calcium and vitamin D deficiencies are found to be predisposing factors. Worms, particularly round worms (ascaris lumbricoides), are looked upon as the most common cause of convulsions. Intestinal toxæmia in a greater or lesser degree is a constant factor and perhaps the most important cause.

In the series mentioned above only five cases were serious, the rest were all simple and three of them were more or less habitual. The simple cases are those in which the only cause of convulsions seem to be temperature. Convulsions are not common below 101°F. temperature. They often occur when the temperature rises above 103°F. The convulsions in these cases are mild. The child has a vacant and fixed look, the whole body goes into a spasm and clonic movements, if they follow, are mild. The duration of the convulsion is short. The abdomen is generally full but not tense and even

during the convulsion the spasm of the skeletal muscles is not very severe. The child is easily roused from this state by smelling Ammon. Carb. The first thing to do is to give a good enema and clear the bowels. This is followed by a good purgative. Calomel and castor oil are the most commonly given purgatives. If the temperature is high it is rapidly brought down by hydrotherapy. Ice bag to the head and cold pack to the extremities. With the temperature maintained below 101°F, convulsions are not likely to recur. Then the child is brought rapidly under Bromides by giving 2·5 gr. of Bromides in a simple diaphoretic mixture, every two to four hours till the child sleeps relaxed. Thereafter it can be continued at longer intervals.

In the severe cases the convulsions are severe and prolonged. The child is blue, the look is fixed and vacant or the eye balls are rolled up. There is severe spasm of the muscles of the body. The abdomen is bloated and tense. Violent clonic movements of the limbs and body follow, including the lips. There may be frothing at the mouth. The child is deeply comatose and insensible, breathing is short and hurried, and the pulse rapid and feeble. If this state is not rapidly relieved death supervenes by respiratory failure. This type of convulsions is probably due to severe intestinal toxæmia. Usually there is a history of constipation and indigestion. It is an urgent necessity to relieve the convulsive state. Bowels should be cleared well. It is always more advantageous to give a copious soap and water enema than a small glycerine enema. Greater elimination of intestinal toxins is to be expected by clearing a greater length of the colon with a water enema than by clearing the lower few inches of the rectum with a glycerine enema. Counter irritation is commonly resorted to in reviving the patient from the coma. The crude indigenous method of branding the face and temples with a live cigar is gradually becoming unpopular though it is still common in the rural areas. Smelling Ammonia Carb. is the simplest thing to do. If that fails, alternate cold and hot baths are very useful. This helps to stimulate the nervous system and is a very useful measure. Besides counter irritation to rouse the patient, something rapidly effective has to be done to steady the nervous system and stop convulsions. The usual thing to do is to give two to three grains of Pot. Bromide by mouth every two hours or more frequently till the child comes under its influence. A Chloral and Bromides retention enema is very useful in rapidly bringing the convulsions under control. Paraldehyde enema is sometimes very effective. But its usefulness is variable. Perhaps Paraldehyde given intramuscularly is more advantageous. Some Barbitone preparations are claimed to give very good results. It is dangerous to give Morphia as it tends to increase respiratory distress. In some of the obstinate cases Mag Sulp I.M. was very helpful and the child relaxed rapidly. Hydrotherapy to control the temperature has to be carried on along with the measures to control the convulsions. When the convulsions have stopped and

the child has relaxed and is restfully sleeping under the Bromides, treatment is directed towards avoiding recurrence of the fits. This resolves itself into eliminating the toxins, toning up the nervous system and reducing the temperature. Free purgation, a simple diaphoretic mixture with Hexamine and Bromides, are all that are necessary. A close watch on the temperature has to be maintained and hydrotherapy resorted to whenever the temperature rises above 102°F. Hexamine is very helpful in combating toxæmia. Drastic and depressing antipyretics are unnecessary and should be avoided. If the temperature persists beyond 48 hours, causes for it other than intestinal toxæmia, particularly malaria, should be investigated. With treatment on the above lines, results have been quite satisfactory. In the five serious cases in our series only one case died. The child had a dose of Perchloride of Mercury before coming to the Nursing Home and she died 48 hours after admission though she was free from convulsions and temperature was well under control for 36 hours. It is a common indigenous practice to give Perchloride of Mercury in these cases and very probably this case failed because of over dosage of the Perchloride.

The three habitual cases referred to were children between the ages of two and seven years. They get convulsions whenever they get the slightest fever for any reason. The fits are light. The child is never completely comatose or unconscious. She is easily roused by smelling Ammon. Carb. and a dose of Calomel with castor oil followed by a diaphorertic mixture with Bromides is all that is necessary for their recovery. Contrary to the common belief, round worms could not be detected in these cases even on repeated examination of the stools. The children are well nourished and healthy looking and do not show any signs of malnutrition or rickets. However, deficiency in calcium and vitamin D is most probably the cause of this extreme predisposition for convulsions in these children.

Stools were not systematically examined in all the cases of this series. However, round worms were found in three, one severe and two light cases. The comparative infrequency of worms may partly be explained by the class of patients that come to a private nursing home. While under the comparatively unbalanced state of the nervous system in a child, worms can be irritation enough to cause convulsions, toxæmia of a more severe type is to be suspected especially in the severe cases. Perhaps mild convulsions with mild general disturbance are more often due to worms than severe convulsions with high fever and deep toxæmia. Calcium and vitamin D are certainly very helpful in building up the child and its nervous system. Deficiency in these can be a decidedly predisposing factor. This deficiency should be suspected in children prone to convulsions even if they do not show obvious signs of the deficiency and the deficiency made good.

ANÆMIA IN INFANCY AND CHILDHOOD

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THE study of anæmias of infancy and childhood requires a preliminary knowledge of the various physiological changes occurring in the blood picture of an infant as it adjusts itself to the extra uterine conditions and demands of growth.

Haemoglobin:—During the first three months of life, the infant shows a sharp drop in the hæmoglobin percentage. The high birth value of 20 gms. percent falls to about 10·5 gms. per-cent. The premature infant shows even still greater fall, the hæmoglobin percentage falling as low as 6·5 gms. percent. This physiological drop cannot be prevented by treatment with iron. From the fourth month onwards, the hæmoglobin begins to rise steadily, reaching the level of 11·5 gms. percent by the end of sixth month. In the second year, with the taking of a mixed diet the hæmoglobin may further rise to the level of 13·0 gms. percent.

Artificially fed infants show 5 to 10 percent lower hæmoglobin levels as the artificial milk is a poor source of iron.

Red cells:—These drop from the birth level of about 6·8 million per c.m.m. to about 5 millions per c.m.m. during the course of the first twelve days. There is a corresponding increase in the icterus index which rises to a peak in three to five days and remains high during the first twelve days of life.

The red cells show a further small fall of about ½ million during the first twelve years of the childhood which adjusts itself later to the adult level of 5 millions by the age of 17 years.

Immature cells:—At birth the peripheral blood of the new-born shows a fair number of nucleated red blood cells, mainly normo-blasts and few erythroblasts, and a fair number of reticulocytes. The premature infant shows more numerous nucleated cells than the full term infant. By the end of the first week the nucleated red cells may fall from the level of 5000 per c.m.m. to the level of 200 per c.m.m. and the reticulocytes may fall from the level of 10% to the level of 1%.

Leucocytes:—After an initial temporary fall following birth the leucocytic count is fairly well maintained in the infant at the level of 17000 to 20000 per c.m.m. This level gradually falls to the adult level of 6000 per c.m.m. by the end of twelve years of life.

During the period of first twelve days the lymphocytes begin to rise from the birth level of about 15% to the level of about 55% and the polymorphs fall from 60% to 25%. In the second phase, which is gradual and prolonged, this differential count adjusts itself to the adult level of 60% polymorphs and 20 to 30% lymphocytes by the end of twelve years.

Bone marrow:—Bone marrow in children shows a large number of immature red cells and lymphocytes. During the first three or four years of life, all bones of the body show red hæmopoietic marrow. From the age of seven years the hæmopoietic activity of the long bones recedes till in the adult it is restricted to the flat bones of the skull, thorax and vertebræ.

Besides bone marrow, extra-medullary erythropoiesis may be observed in liver, spleen and pelvis of the kidney in both premature and full term infants upto the period of first fifteen days and later these centres may again assume blood-forming activity if required, *e.g.* in severe anæmia.

Platelets:—Infants show the same count of platelets as the adult.

Coagulation time:—The infant has a low prothrombin content normally. This level of prothrombin falls further in the next forty-eight hours and gradually reaches the birth level in about five days. During this period the intestinal bacterial flora is inadequate to synthesize vitamin K which is normally converted into prothrombin by liver. Hence there is a deficiency. The coagulation time, therefore, is higher by about 20% to 40% during the first five days of life.

Fragility of the red blood cells:—There is no definite evidence of increase in the fragility of red blood cells in the new born.

Iso agglutinins:—Infants are born with fully developed agglutinogens but they usually contain agglutinins derived from the mother. Child's own agglutinins begin to appear from the tenth day of life, as such maternal blood can be safely transferred during the first ten days without compatibility tests.

Gastric function:—Gastric acidity is lower in infants than in older children. Infection of the alimentary tract tends to decrease the acidity.

Normal erythropoiesis:—Embryonic studies show that blood cells arise from the area vasculosa of the yolk sac. Early in fœtal life the spleen and liver take over this function, to be replaced later on by the bone marrow. From the fifth month of fœtal life the red cells are formed from the endothelial linings of the blood vessels in these tissues. The primitive hæmocytoblast develops into the megaloblast, the erythroblast, the normoblast, the reticulocyte and the erythrocyte (normal red blood cell). During this process, the cells diminish in size at each stage of maturation and begin to acquire hæmoglobin from the stage of erythroblast.

Castle's hæmopoietic principle is required for the proper maturation of the red cells. This principle is formed by the interaction of a gastric enzyme (intrinsic factor) and the extrinsic factor derived from the protein of the diet. The deficiency of this substance will produce macrocytic anæmia. The fœtus derives this principle from

the mother in intrauterine life. Later on, it manufactures its own requirement.

Thyroxine, vitamin C, traces of copper and manganese are also essential for the perfect production of the red cells.

The mature red cells live for a period of about thirty days. They are then broken down by reticulo-endothelial cells and the iron liberated is mostly retained for further manufacture of hæmoglobin, though some is lost through excretion.

The colour index is the numerical expression of the hæmoglobin content of the red cells. The colour index is less than one if the hæmoglobin is reduced to a greater extent than the number of red cells. On the other hand, the colour index is greater than one, if the red cells are greatly reduced without corresponding decrease in the hæmoglobin.

Types of anaemia.—By means of red cell size and mean hæmoglobin content anæmias are classified on a basis which gives an indication of the nature of the disorder. The terms 'hypochromic' 'normochromic' and 'hyperchromic' indicate whether the cells contain an amount of hæmoglobin which is less than, equal to, or more than normal, and the terms 'microcytic', 'normocytic' and 'macrocytic' suggest whether the cells are smaller than, equal to, or larger than normal.

Of all the theoretically possible permutations, the commonest are:—

(1) Hypochromic normocytic anæmia.
(2) Hypochromic microcytic anæmia.
(3) Hypochromic macrocytic anæmia.
(4) Hyperchromic macrocytic anæmia.

The term 'hyperchromic' is misleading because it implies over-saturation. Concentration of hæmoglobin in a normal red cell and a macrocyte is the same but the fully hæmoglobinised macrocyte contains more hæmoglobin than a normocyte by reason of its size. Hence the colour index is greater than one.

The causes of the various types of anæmias may be classified as follows:—

I. Deficiency of materials needed for the production of red cells:—(1) Deficiency of iron. (2) Deficiency of hæmopoietic principle. (3) Deficiency of vitamins and endocrines.

II. Hypoplasia and aplasia of the bone marrow.

III. Loss of the finished product:—(1) By hæmorrhage. (2) By hæmolysis.

I. Deficiency anæmias.—1. *Iron deficiency anaemia*:—Primary iron deficiency anæmia is observed in young babies from the ages of three months to six months, particularly in those who

are artificially fed or prematurely born. Liver stores iron derived from the mother during the last few months of pregnancy and the store is augmented by the iron from the physiological hæmolysis of the red cells in the new-born. However this store is soon depleted during the first few months of life. Milk, particularly the artificial one, is a poor source of iron and as such babies who are artificially fed, or those who do not get fully sufficient breast-feeds, or those born of anæmic mothers are likely to develop this type of anæmia. Anæmia of twin births is also due to the same cause. In the premature baby there is an additional factor of excessive blood destruction which aggravates the condition.

The anæmia is hypochromic, normocytic or microcytic.

Similar anæmia may also be observed in babies past milk feeding period, particularly amongst the poorer classes unable to purchase expensive iron containing mixed diet.

Secondary iron deficiency anæmia is seen in later childhood with chronic disorders of the gastro-intestinal tract like cœliac disease, helminthiasis etc.

Similar hypochromic anæmia may also be the result of debility and malnutrition following acute rheumatic fever, tonsillitis, malaria and other serious illnesses.

TREATMENT.—Intensive iron treatment is usually successful except in premature babies who should be well protected from infection and given a diet rich in vitamins and mineral salts.

In the secondary type, proper treatment of the primary disorder is necessary in addition to the iron therapy.

2. *Deficiency of haematinic factor.*—True pernicious anæmia is extremely rare in infancy and childhood. However, macrocytic anæmia following gastro-intestinal conditions with lack of absorption of hæmatinic factor may be observed occasionally. Usual treatment with Folic Acid, Vitamin B_{12} or Liver Extract is advocated.

3. *Deficiency of vitamins and endocrines*:—Scurvy manifested by bleeding in joints, subperiosteal spaces and mucous membranes shows hypochromic anæmia, which may appear even before the scorbutic symptoms.

Rickets is usually associated with von Jaksch's syndrome and the anæmia is hypochromic.

Cretinism and hypothyroidism shows macrocytic hypochromic anæmia and responds to thyroid extract.

II. Dysfunction of the bone marrow.—Bacterial toxins and chemical poisons cause hypoplasia and aplasia of the bone marrow depressing the function of red cell formation. Typhoid fever, diphtheria, bacterial endocarditis and latent tuberculosis are common causes of this type of anæmia. Similarly, arsenical therapy, sulpha drugs and lead poisoning from sucking of toys and furniture painted

with colours containing lead may produce hypoplasia of the bone marrow. Urea retention of chronic nephritis has also a similar effect (Shelle, 1945).

Radium and X-ray therapy for malignancy or skin diseases may produce similar depression of the function of the bone marrow.

Bone marrow, on the other hand, may itself be destroyed by deposits of lymphosarcoma, leukæmia and other tumours.

The anæmia in these cases is usually severe and the count reaching as low a figure as 1 million per c.m.m. Hæmoglobin is also proportionately reduced. Hence the anæmia is normocytic, normochromic.

Treatment consists in dealing with the cause. Repeated blood transfusion and iron therapy may be necessary.

III. **A.** Anæmias of hæmorrhage.—1. Acute or chronic blood loss may be observed in injuries, operative procedures like tonsillectomy, and in polypoidal condition of nose or rectum.

Acute hæmorrhagic nephritis may also result in severe blood loss. The type of anæmia is usually microcytic hypochromic.

Treatment consists of dealing with the cause of hæmorrhage, blood transfusion if there is excessive loss of blood, and iron therapy to supplement the same.

2. *Haemorrhagic diathesis or melaena neonatorum*:—The infant is normally born with a low plasma prothrombin. This level of prothrombin falls further in the next fortyeight hours and reaches the birth level in about five days. During this time the intestinal bacterial flora is inadequate to synthesize vitamin K which is normally converted into prothrombin by the liver. Hence there is a deficiency. This deficiency may be marked to produce the hæmorrhagic diathesis. However, it must be stressed that prothrombin deficiency does not afford a complete explanation for the condition. There is also associated deficiency of thrombokinase and fibrinogen. Hence bleeding time, prothrombin time and the coagulation time are all prolonged. Coagulation time may be prolonged three or twelve times the normal.

The bleeding usually starts suddenly between the first and the fifth day of life. It reveals itself by melæna or hæmatemesis. At times, the bleeding may occur at the umbilicus or at the various mucous surfaces. Internal hæmorrhage is likely to be mistaken as due to a birth injury. Cessation of bleeding is also sudden within the first week if the child revives.

This differentiates the condition from hæmophilia where there is persistent bleeding. Test for occult blood in stool is deceptive. The positive result may be due to blood swallowed by the baby during birth from the cracked nipples of the mother. Examination of bleeding time, coagulation time and prothrombin time are the proper laboratory procedures for the diagnosis.

Prognosis depends upon the weight of the child, amount of blood lost and secondary infection.

Treatment consists of adequate doses of vitamin K, blood transfusion and intramuscular injection of 10 to 20 cc. of maternal blood.

3. *Haemophilia*:—This condition differs from the above in that the bleeding does not occur at birth or within the first few months of life. It usually starts later, the bleeding is persistent and dangerous. Bleeding into and round about joints may simulate rheumatism and occurs after trivial injuries. The anæmia is hypochromic with low colour index. The coagulation time is markedly delayed.

The condition is supposed to be due to an unidentified substance in blood which interferes with the breakdown of platelets to release thrombokinase. It is a hereditary condition and transmitted by females to males.

Treatment consists of local application of pressure and coagulants. Blood transfusion is usually required.

4. *Purpura haemorrhagica*:—This condition is rare in infancy. Usually it is preceded by infection and hence it is secondary. Spontaneous remission may occur (Talmadge and Bermain, 1947). Transfusion helps recovery. Splenectomy has high mortality and is not always successful in ameliorating the condition.

B. Hæmolytic anæmia.—1. *Haemolytic disease of the newborn*:—This condition is usually the result of Rh anti-bodies in the blood of an Rh-negative mother formed against the Rh-positive cells of the fœtus. These anti-bodies pass from the maternal blood to fœtal circulation and cause hæmolysis of the cells of the Rh-positive fœtus. If the father is Rh-positive and the mother is Rh-negative the offspring will be Rh-positive in 100 per cent of the pregnancies, if the father is homozygos and Rh-factor is inherited as a dominant trait. If he is heterozygous only 50% of offspring will be Rh-positive. It is estimated that 85% of general white population are Rh-positive, 95% of the coloured race and 99·2% of the Oriental (Sanford, 1950). Incidence of the hæmolytic disease, however, is not so common as compared with the possible chances of unions of Rh-positive father and Rh-positive mother. The reason is that Rh-negative mothers are not always capable of manufacturing these anti-bodies and fœtal blood must get through the placental barrier into the maternal circulation. Usually the primigravida does not develop sufficient anti-bodies to produce hæmolytic disease (Weiner and Hallum, 1950). However, the multipara or the mother with previous abortion or transfusion with Rh-positive blood or even intramuscular injection of Rh-positive blood, may develop these anti-bodies in high proportion (Levine, 1941). In a small proportion of cases the hæmolytic disease is produced by atypical Rh anti-bodies or by potent anti-A or anti-B agglutinins (Gruber *et al*, 1946) antagonistic to the fœtal cells.

Three distinct manifestations of this condition are recognised: (*a*) Hydrops fœtalis; (*b*) icterus gravis neonatorum; and (*c*) anæmia gravis neonatorum.

(*a*) Hydrops fœtalis is a generalised œdematous condition of the fœtus, usually still-born or dying within a few hours after birth, associated with effusions in serous cavities, splenomegaly and hepatomegaly and enlargement of heart. There is usually a severe anæmia with count as low as ½ million with nucleated cells which may be as high as 60%. A slight jaundice may also be present.

(*b*) Icterus gravis neonatorum is a condition of severe jaundice and anæmia often familial, with a high degree of mortality of 50% to 80%. Some of the survivors develop central nervous system lesions like spasticity, athetosis, or mental deficiency due to toxic degeneration of brain cells by bile pigments (kernicterus). Vaughan suggests that blood destruction is not the primary determinant of nuclear damage.

The infant shows jaundice from birth or shortly afterwards which lasts for about a week or two and later fades, leaving a very pale child. Indirect Van den Bergh reaction is positive though the test may become diphasic later due to destruction of liver cells. Stools are normal and there is excess of urobilin in urine.

Hæmorrhages at the umbilicus and the various mucous membranes may also be observed.

The anæmia is usually severe and macrocytic in type with large number of erythroblasts and a fair number of reticulocytes.

Hepato-lenticular degeneration (Wilson's disease), Banti's disease and juvenile cirrhosis are considered to be the complications of this condition.

It is important to distinguish this condition from other conditions giving rise to jaundice in the new-born. Simple physiological jaundice which is usually noticed on the third day clears up in about three days and is not associated with splenomegaly. Severe infective jaundice is associated with grave signs of toxæmia, fever hæmorrhages etc. with primary cutaneous infection, intestinal infection or umbilicus sepsis. Congenital obliteration of the bile ducts is associated with splenomegaly and hepatomegaly and jaundice is much more prolonged and stools are clay coloured owing to the absence of bile. Acholuric jaundice is rarely seen in the new-born and not earlier than the second week of life. Fragility test will clear the diagnosis.

(*c*) *Anaemia gravis neonatorum*:—In this condition there is a severe anæmia without icterus. The condition is usually seen at the end of the first week but may be delayed for one or two months with a milder attack. Fair number of immature cells mainly consisting of erythroblasts are seen together with increased number of reticulocytes. Progress of anæmia is fairly rapid with complication of an intercurrent infection.

TREATMENT.—Prophylactic treatment of the mother with vitamin C to strengthen capillary permeability of placental villi, injection of Carter's protein-free haptene prepared from Rh-positive cells to neutralise Rh antibodies, and even premature labour to relieve fœtus of its pernicious environment are advocated. Wolf *et al* (1950) however report that Carter's haptene is not of any clinical value in altering fœtal prognosis.

Active treatment consists of transfusion, preferably of Rh-negative blood, though Rh-positive blood can also be used (Sanford, 1950). Mayes (1946) reports successful treatment even with mother's blood.

Substitution transfusions have come into vogue recently. They are particularly useful in serious cases. Oxygen therapy is also helpful.

Mother's own milk should not be given for at least the first two weeks as Rh anti-bodies are present in it, especially in the colostrum. Carter's haptene may also be tried.

2. *Lederer's anaemia*:—This is an acute hæmolytic anæmia with fever occurring in infancy and childhood between the ages of six months and three years. The onset is sudden with an attack of jaundice, fever and hæmaturia. The anæmia is severe, macrocytic in type and associated with marked leucocytosis. Reticulocytes together with immature white blood cells like myelocytes and metamyelocytes may be seen. The count may be as low as 1 million per c.m.m. There is no increase in the fragility of the red cells.

The spleen and liver usually enlarge rapidly and there are gastro-intestinal symptoms like vomiting and diarrhœa.

No specific cause for this condition has been detected though an infective focus is generally blamed. Another explanation given is an incompatibility of the Rh subgroup in both Rh-positive mother and fœtus. Sudden onset may be due to some obscure precipitating cause which also explains the delayed hæmolysis.

TREATMENT.—Early transfusion has a dramatic effect in ameliorating the condition which otherwise is likely to pass into aplastic phase. Vigorous iron treatment may also be needed.

3. *Congenital haemolytic jaundice or acholuric jaundice*:—This condition is rarely seen in the new-born but may be met with in later childhood. It is a familial disease associated with a congenital defect of the erythron with production of small globular red blood cells (microspherocytes) with characteristic disease in fragility.

It is characterised by recurrent attacks of jaundice due to repeated crisis of blood destruction. The urine is free from bilirubin and the urobilin excretion in urine may be as high as 10 to 30 times normal. The spleen is generally enlarged and tender and may reach the umbilicus. Liver is also enlarged and tender.

The count may be reduced to about 1 million per c.m.m. The icterus index is high and Van den Bergh test is indirect positive. The fragility test shows hæmolysis beginning with 0·75% and completing in 0·4% solution of salt. The red cells are spheroidal in shape and fair number of normoblasts and reticulocytes upto 50% may be seen. Fragility test may be normal in between the crisis. The crisis may be precipitated by fatigue or infection.

Treatment consists of splenectomy in between the crisis. Alternatively, ligation of splenic artery and X-ray treatment of splenic area may be useful. Blood transfusion is useful after splenectomy.

4. *Sickle cell anaemia*:—This is a hereditary and familial condition due to blood dyscrasia of the Negroes characterised by severe hæmolytic anæmia with a peculiar sickle shape of the red cell appearing under lowered oxygen tension. The spleen is enlarged but the fragility of the red cells is not increased. There is jaundice with raised icterus index and indirect positive Van den Bergh reaction.

The count is usually 1 to 3 million with proportionate reduction in hæmoglobin and the colour index is 1·0. Leucocytosis is always present. Fair number of reticulocytes, as high as 25%, may be seen. The normoblasts are also seen.

Treatment consists of continued oxygen inhalation. Splenectomy and blood transfusion may be useful in selected cases.

5. *Cooly's anaemia*:—This condition is a chronic progressive hæmolytic anæmia commencing in early life from six months to three years with characteristic erythroblastosis in peripheral blood, typical Mongolian facies with thickening of cranial bones and molar eminences, muddy discolouration of skin and splenomegaly. It is a familial disease endemic in the Mediterranean area with a fundamental defect of the red cells which are usually thin, large and appearing almost as colourless membrane with a rim of hæmoglobin at the periphery—the so-called 'Target' cells. There is usually an associated leucocytosis.

The icterus index is raised and the Van den Bergh test is indirect positive. Urine contains excess of urobilin. The fragility of the red cells is normal. A few cases of this condition have been observed in India (Dhayagude, 1944, Chandra, 1951).

TREATMENT.—No treatment is satisfactory, splenectomy seems to prolong life. Govan reports strikingly good effect of splenectomy in twins suffering from Cooly's anæmia.

6. *Haemolytic anaemia in toxaemia*:—Various toxins, bacterial or chemical, such as toxins of streptococcus hæmolyticus, sulpha drugs, arsenicals may produce severe hæmolytic anæmia. Malaria parasites destroy the red blood cell directly. Black water

fever, though rare in children, is due to the excessive destruction of the red cells.

Treatment consists in dealing adequately with the cause of the condition.

Von Jaksch's syndrome.—Von Jaksch described a chronic hypochromic microcytic anæmia commonly seen in children between the ages of six months and three years with splenomegaly, moderate hepatomegaly and enlargement of lymph glands. There is usually associated lymphocytosis. The syndrome is usually accompanied with rickets or chronic infective process, congenital syphilis, or malnutrition. Thus it will be seen that it can hardly be considered as a separate disease entity. Iron deficiency, chronic hæmolysin and depressed marrow functions are the three ætiological factors.

Treatment depends upon dealing with the cause complicating the condition. Intensive iron therapy and transfusions may be necessary.

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DYSTROPHIA ADIPOSOGENITALIS

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DYSTROPHIA adiposogenitalis or Frohlich's Syndrome* as the disease is commonly termed after the name of the medical scientist who observed and reported a typical case just fifty years back, is not at all an uncommon disease in childhood. As the disease is not usually diagnosed unless the patient has stepped into his or her teens it is supposed to be a disease of the adolescent period but a slow and insidious onset must have taken place much earlier in life during the childhood. It takes months and sometimes years for the typical syndrome with the three essential triad, *viz.*, retardation of skeleton, undeveloped sexual organs and obesity to develop, which facilitates the correct diagnosis and so the infantile or the pre-pubertal type is considered as rare simply because the symptoms are either latent or do not make their appearance concurrently in an earlier period of life. But when a few years hence the symptoms, although slow and imperceptible in the beginning, manifest themselves clearly in an adolescent or a young adult, the condition very often baffles treatment and chances of cure are few and far between. So a diagnosis and commencement of the treatment as early as possible are essential for the prospect of a complete cure.

Historical

In 1900 Babynsky reported the earliest case of a girl with obesity, sexual immaturity and enlargement of the sella turcica. One year later on Frohlich published the case note of a boy aged 13, who suddenly developed severe frontal headache followed by vomiting. This boy who was rather slim at this time in the course of the next two years gained in weight rapidly with diminution in the sight of the left eye and subsequently of the right eye as well. The height of the boy was almost normal for his age but he was definitely overweight due to excessive deposition of fat in the abdomen and in the neighbourhood of the genitals which were undeveloped, and his general appearance was infantile and feminine. So originally Frohlich's Syndrome was a symptom complex presenting a

* In the course of the last four years the writer has detected the syndrome in eleven children between the ages of three and ten, seven being males and four females. Enlargement of the sella turcica was found in one girl and that case alone did not respond to medical treatment. Of the seven male children five have responded to treatment lasting for three to six months so that their growth, development and appearance have become quite normal again, while two others are still undergoing treatment. Except the case of the girl mentioned already, another girl unluckily died of severe burns during the course of treatment while the other two have grown into fine normal adolescent girls after a course of treatment.

typical picture of concurrent infantilism (without dwarfism), obesity and diminished vision due to a tumour of the pituitary gland or in the neighbourhood. In 1906 Bartel styled the syndrome as dystrophia adiposogenitalis. Later on Frohlich reported other cases, one of which was operated on by Eiselberg in 1907 and a cyst of the size of a hazel nut was removed by the nasal route with definite improvement in general condition and eye sight and occurrence of less headache, and a year later there was cessation of headache and giddiness and slight sexual development. The same case was again reported by Biedl in 1913 as 'Biedl-Moon-Syndrome', when the general improvement continued but infantile habits and obesity were still present[1].

AETIOLOGY.—According to Frohlich the disease is due to a tumour of the pituitary or in the neighbourhood but according to Eiselberg (1907) von. Frankl-Hchwart (1908) and Fulton and Bailey (1929), the obesity was not due to a pituitary-tumour but probably due to a lesion in the hypothalamus nearby. This has been confirmed later on by Smith who showed by experiments that injury to the hypothalamic region of the brain caused a marked genital atrophy with obesity without affecting the thyroid and the adrenal cortex but hypophysectomy in young rats resulted in atrophy of the genital system, the thyroid and the adrenals without obesity[2]. So the disease may be caused either by an inherent defect in the hypophysis (may be due to a tumour of the gland itself or atrophy of the secreting cells by pressure due to growths in the neighbourhood) or by a lesion in the hypothalamo-hypophysial nervous path owing to an injury due to a penetrating wound or as a result of some infectious disease. Along with the dysfunction of the pars anterior of the pituitary there is also noticed some hypofunction of the thyroid, adrenal cortex and the gonads as the first being 'the leader of the endocrine orchestra' by virtue of its thyrotrophin, adreno-corticotrophin and gonadotrophins respectively directly controls the functions of the thyroid, adrenal cortex and the sex glands and through the thyroid hormone indirectly also affects the gonads. Besides, by two other hormones, the metabolic as also the adreno-corticotrophic hormones (A.C.T.H.), it also regulates to a considerable degree the body metabolism in general. So although the pituitary gland is mainly responsible for the causation of this condition, dysfunction of the three other subsidiary endocrine glands more or less, is also present side by side and hence the causal factors and the course of the disease are much more complex and complicated than hitherto supposed.

According to some[3] the onset of the disease commonly occurs between the ages of eight and ten, when it is comparatively sudden with marked and rapid increase in weight accompanied by slowing of the skeletal growth. But to the experienced eyes of an endocrinologist the syndrome could be detected at any age before puberty,

specially from three to ten years of age. As a rule the earlier the onset the greater is the degree of stunting of the skeleton but dwarfism which forms one of the essential symptoms in Frohlich's original description is by no means an invariable accompaniment of the disease, as sometimes cases are noticed with either normal or even supranormal skeletal growth.

SYMPTOMS AND SIGNS.—In male children there is unnatural obesity with deposits of fat with a feminine type of distribution producing large hips, abdomen, thighs, upper arms and mons veneris. The penis is small, the testes are underdeveloped and sometimes undescended.

In the female child there is marked fattiness, the skin is soft and pale and the hair is fine and silky. The external genitalia are small and infantile and the breasts become big early, not due to glandular development as usually occurs during puberty but as a result of padding of fat in that region. The 'high hip' is caused by unusual fat deposition on the lower girdle together with similar well marked deposits on the subscapular and nuchal areas.

In both the sexes, the eyes are sleepy, the face as also rather thin legs and forearms are graceful, the hands and feet are small and tender, and the fingers are delicate, narrow and tapering. The skin is soft, glossy and hairless except over the scalp where it is fine and silky. The knock-kneed stance is indeed very, very characteristic. The patients are commonly musical, imaginative and asthetic in their tastes and the intelligence is either normal or below par. They are, as a rule, very much liked by their friends and relations not only because of their graceful appearance but also for their excellent demeanour and genial temperament. Sometimes there are periods of joy alternating with those of depression. In well advanced cases patients may complain of persistent headache, giddiness, faintness and failing vision. They are usually very, very fond of sweets and other delicacies with increased carbohydrate tolerance and decreased sensitivity to Insulin and Adrenalin. The general metabolism, the B.M.R. as also the specific dynamic action of proteins, are all depressed.

DIAGNOSIS.—For an early diagnosis of the condition some bodily measurements are of great value: (*a*) The body-weight is much in excess in comparison to that of a normal child of the same age and height.

(*b*) The standing height is exceeded by the span (which should be the same in a normal person).

(*c*) The lower measurement (from symphysis pubis to the foot) is less than the upper (from vertex to the symphysis) which should be equal.

(*d*) Measurement of the abdomen at the umbilicus is a few inches more than the circumference of the chest in the nipple line

in a male or just below the breasts in a female (which should normally be the reverse).

(*e*) The circumference of the head is sometimes greater than the normal. Besides, X-ray skiagrams showing expansion of the sella turcica and nonclosure of the epiphyses of certain bones[4] in their proper times are also of assistance in arriving at a correct diagnosis. These valuable signs together with infantile genital organs, characteristic deposits of fat in the abdomen, breast, thighs, upper arms, nuchal and subscapular areas together with other characteristics of the different parts of the body must confirm the diagnosis. Moreover, increased carbohydrate tolerance, diminished sensitivity to Insulin and Adrenalin and lowered B.M.R. should facilitate an early diagnosis. But it should always be borne in mind that simple adiposity in children without any physical defects may be due to sedentary habits, voracious appetite, gluttony, parental attitude and environmental factors ([5, 6 and 7]) which must be thoroughly excluded before coming to the diagnosis of this condition.

PROGNOSIS.—This depends greatly upon the site and extent of the lesion as also upon the affection of the various glands. Lesions caused by injuries to the hypothalamus hypophysial nervous path, tumour of the pituitary gland or of the neighbouring structures augur a bad prognosis, and cases due to hypoplasia of the hypophysis or involvement of the hypothalamic region due to a mild infection have got a better prognosis as regards life but the patients may grow and live with a handicap (feminine appearance, womanly voice, lack of beard and mustache and impaired sexual functions in the males and amenorrhœa or scanty menstruation, infantile uterus and consequently sterility in females) and due to unusual obesity they stand the risk of intercurrent infection which may be responsible for their early or premature death.

An early diagnosis and prompt treatment are absolutely necessary for a complete cure in a majority of cases and relief of symptoms in others.

TREATMENT.—Restriction of diet and regular physical exercise are of primary importance. In fact, if the condition is diagnosed early with the onset of the disease, a strict restriction of the diet for about six months with some especially recommended physical exercise have been found very, very useful in many cases. The diet must consist of proteins in plenty, fruits and green and leafy vegetables with restriction of carbohydrate to the irreducible minimum and complete exclusion of fat. The daily caloric intake must not exceed 2,000 calories in the beginning. As usually there is much craving for sweets and other fattening delicacies these must be scrupulously avoided and the voracious appetite ought to be cut down by drugs like Dexedrine or Benzedrine (one tablet b.d.s. or t.d.s. during the first half of the day) which will also prevent sleep during day time, increase the body metabolism in general, and help to counteract somnolence and lethargy due to hypothalamic lesion.

These drugs must not be given in the afternoon or at night as they may cause sleeplessness during the night.

In some cases a fat-reducing diet consisting of milk and banana only have been found to be an ideal diet and a very efficacious adjunct to the specific treatment.

Bearing in mind that dystrophia adiposogenitalis is a complex pluriglandular dysfunction, the endocrine therapy should include not only the correction of the pituitary condition but also that of the hypothyroid and hypogonadal conditions. Along with the regular physical exercise and a low caloric suitable diet, desiccated thyroid in doses tolerable does good long before the patients have reached the typical, and to some extent therapeutically refractory, stage. For this the B.M.R. and Cholesterol content of the blood must provide a valuable guidance. For gonadal maturation Chorionic Gonadotrophins like Antuitrin-S or Synapoidin etc. are of great value, which help to set right the pituitary dysfunction and have also been found beneficial in some cases of cryptorchidism (without organic obstruction or defect) by promoting descent of the undescended testis into the scrotum[8]. The phallic growth as also the development of the secondary sex characters (in both sexes) are also materially helped by these preparations. For the development of the small sized testes as also that of the penis sometimes Methyl Testosterone by mouth or Testosterone Propionate by injection is also helpful. The development of the utero-vaginal tract and acceleration of the secondary sex characters in girls require the administration of œstrogenic hormones like Theelin, Ovocyclin, Lutocyclin etc. But one has to be very very careful and discreet in using these hormonal preparations as, if used injudiciously, like a "double barrelled gun" well loaded, they may do more harm than good.

Surgical intervention is necessary for all those cases due to pressure within the sella turcica causing intense and persistent headache, projectile vomiting, gradually failing vision, first in one eye and then in both, as also those due to a lesion in the hypothalamo-hypophysial tracts by an injury. According to Cushing and Henderson a successful surgical measure in time restores not only the sight but also the impaired sexual functions ([9 and 10]). In inoperable cases, or when the patient is reluctant to have an operation, deep X-ray therapy might sometimes give encouraging results.[11]

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MUSCULAR DYSTROPHY

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THESE are a group of disorders in which the essential lesion is a progressive degeneration of certain group muscles. The disease is familial or hereditary and various types have been described according to the involvement of the groups of muscles, so the muscular dystrophies have several clinical lesions but the basic pathology remains the same. Nothing is known regarding the cause of this condition but there is a hereditary fault. What that fault is, we do not know. It appears that like hæmophilia, it is transmitted by healthy females and occurs usually in males.

PATHOLOGY.—In certain cases there is atrophy as well as pseudo-hypertrophy producing a marked enlargement of certain muscles. Although the muscles enlarge they are without power. "All is not gold that glitters." According to Buzzard and Greenfield there is a swelling and increase of the sarcolemma nuclei. After sometime the muscle fibres undergo degeneration and conversion into fibrous tissue. The connective tissue septa are increased between the fibres and great deposits of fat occur between the muscle fibres. The pseudo-hypertrophy and enlargement are due to these fatty deposits. The exact cause for the fatty deposits is not known. Is it due to the metabolic disorder, or is it due to endocrinal disorder? The question is still unanswered. Let us now discuss the various clinical types :—

Pseudo-hypertrophic muscular dystrophy of Duchenne.—This occurs in children. The males are commonly affected than females in the proportion 5 to 1. A child which was previously normal, begins to walk clumsily and falls easily and after falling finds it very difficult to get up without aid. He cannot skip or jump and finds it very difficult in climbing up-stairs. On examination it is found that certain muscles may exhibit conspicuous enlargement like the calfs, the glutei, the infra spinati and the deltoids. At the same time other groups of muscles show atrophy as pectoralis major and the latissimus dorsi serratus magnus.

In the muscles of the limbs the proximal muscles waste more than distal muscles. By this certain pictures are noticed ; in standing the legs are placed far apart ; the upper part of the trunk is thrown backwards so that a plumbline from the vertebra lies far behind the sacrum, as if a pregnant lady is standing. When the child is made to sit the lordosis disappears. When the child is asked to lie down the lordosis appears again but can be abolished by relaxing the flexors of the hip joint. The child is asked to get up from the supine position to erect position, he first tries to sit up but fails. He has to roll over on to his belly and rises himself first on his knees and elbows and then hands and feet. Next he places his hands on the knees and works his way up his thighs in the characteristic climbing movement. To climb the thighs successfully a certain

amount of power is necessary to hold the knees slightly flexed. When this power is lost he cannot rise. Due to the wasting of the pectoralis major, serratus magnus, biceps and triceps producing winging of the scapula and loose shoulders, the child, when lifted by the axilla, will slip through the hands.

The condition is progressive, producing an increase in the weakness of the muscles. Few patients reach adult life and most people die within ten years of the onset of the disease.

Other types.—1. *In the Leyden-Mobius type*, pseudohypertrophy is absent and the wasting predominates in the lower limbs.

2. *The facio-scapulo-humeral type*:—*Dystrophy of Landouzy Dejerine*:—Occurs in childhood. Weakness and wasting first appear in the facial muscles. The weakness of the orbicularis oris renders pouting and whistling impossible. The zygomatic suffer early causing weakness and retraction of the angle of the mouth, which is conspicuous on smiling. Atrophy next involves scapulo-humeral muscles.

3. *The juvenile dystrophy of Erb*:—This disease occurs between the ages of 15 and 35 and wasting occurs in the muscles of the shoulder and pelvic girdle.

4. Gowers describes a *distal type* in which the wasting begins in the forearms and hands and the legs.

5. Nevin describes a *late type* occurring after middle age.

PROGNOSIS.—The pseudo-hypertrophic type is invariably progressive. The disease usually terminates fatally from 10 to 15 years after onset. In the other varieties the disease is less progressive and in some cases the disease is arrested.

DIAGNOSIS.—Anterior poliomyelitis is characterised by acute onset and the asymmetrical distribution of the muscular wasting. Progressive muscular atrophy develops later in life and muscular fibrillation is always present.

TREATMENT:—At the neurological clinic of the Erskine Hospital we had the opportunity of getting a number of cases of muscular dystrophy. During the years of 1949–'50 there were 9 cases of pseudo-hypertrophic muscular dystrophy, out of which 8 were boys and one was a girl. There were two cases of Erb's Juvenile type and one case of facio-scapulo-humeral Dystrophy of Landouzy and Dejerine. 3 cases of pseudo-hypertrophic muscular dystrophy were taken for treatment and progress was made with Glycine 10 grams b.d. and the Tocopherol (Vitamin E) 30 mgm. daily for 4 months. These children came with inability to get up and were able to have greater amount of mobility after treatment. One of the cases was able to walk freely and was shown at the Clinical Society meeting.

Dystrophia myotonica.—The disease is a type of muscular dystrophy which is of familial incidence. The atrophy begins in the sternomastoid and facial muscles and spreads over the forearm, muscles of mastication, the vasti, the dorsi flexors of the feet and the peroneal muscles. Associated with this wasting there is a peculiar

muscular weakness or myotonia which produces difficulty in relaxing the muscles after contraction.

CAUSE.—No causal factors are known. This is one of the most mysterious hereditary disorders. In a family or several generations one generation may get this disease while several generations may be free.

SYMPTOMS.—The onset is gradual. The first symptom to call attention is a difficulty in relaxing after muscular effort. In other words, there is a prolonged contraction and the relaxation is very, very slow. Supposing such a patient is asked to shake hands he is unable to disengage the hand, which he still holds. He can smile and the face remains in the same position for some time. If he opens his mouth, the mouth will remain in that position for some time and he can only close after some time. The age incidence is about 10 years, but I have seen a case of a male patient, aged 25. This patient, a school teacher, came with a history of wasting of the shoulder girdle and weakness of the orbicularis oculi. When he was asked to shut his eyes, he could not open with ease. He had drooping eyelids.

The second feature of the disease is a muscular wasting. This is usually seen first in the facial muscles, sternomastoids and then it spreads over the shoulder girdle, in the forearms and hands, the quadricap muscles and the muscles of the legs below the knees. Pseudo-hypertrophy is rare, fibrillation is absent. A case of dystrophia myotonica can easily be recognised by the mask-like expressionless face, drooping eye lids and sunken cheeks. The reflexes are generally lost or diminished. The voice is low and has nasal twang. The wasted muscles show reaction of degeneration.

Cataract is generally associated with this disease. Vision may be impaired. The testes or the vas are atrophied with a loss of sexual desire. Amenorrhœa occurs in the female. Intelligence is below par.

DIAGNOSIS.—The diagnosis is characterised by such symptoms as muscular wasting, myotonia and other dystrophies.

PROGNOSIS.—It is a slow progressive disease.

TREATMENT.—Russal and Stedman in 1936 suggested that the myotonia is due to the disturbance of function of the myoneural junction, since the symptoms are intensified by the Acetyl Choline, Prostigmin and Potassium and diminished by Quinine. Myotonia is relieved by Quinine Hydrochloride in a dose of 10 grains 3 times daily. There is no known treatment to arrest the progress of the disease.

Myotonia congenita or Thomsen's disease.—This disease is characterised by: (1) Profound muscular weakness; (2) mental impairment; (3) a prolongation of muscular contraction with slow relaxation; and (4) hypertrophy of certain groups of muscles. No cure is known to this condition. Thomsen first recommended vigorous exercise but this appears to do more harm than good.

BRONCHO-PNEUMONIA IN CHILDREN

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THE catarrh of the bronchial tubes that exist in bronchitis until it reaches their smallest branches, and after that the lung cells themselves, is called broncho-pneumonia. The disease is very common in infants upto the age of two years, especially after measles and whooping cough and diarrhœa.

The symptoms are those of bronchitis, only more exaggerated. Restlessness is marked, breathing rapid and obstructed and the alæ nasi working with each breath. The lips become blue and the temperature is usually higher than in bronchitis. The fever may last several weeks and then gradually subsides. But if the child grows worse, he usually becomes drowsy and at length dies of exhaustion. Broncho-pneumonia is a very serious disease, and among small infants quite half of those attacked succumb to it. Croupy pneumonia begins suddenly, from a condition of perfect health, with high temperature which usually persists as continued fever until the crisis comes, possibly also with vomiting. A tormenting irritative moaning pain and retardation of one side of the thorax with breathing show that the lung is affected even though the findings are still negative in the first few days. The onset, however, frequently resembles appendicitis (abdominal pain on the right side, fairly far down, vomiting etc). In every case of uncertain appendicitis the lungs should therefore be X-rayed before operation. Leucocytosis of over 30,000 speaks for pneumonia, of round about 18,000 for appendicitis. If there is suspicion of meningitis or encephalitis, lumbar puncture decides the question; if typhoid fever is suspected, the decision depends on screening, leucocyte counts, etc. Critical defervescence may occur after 5–6 days, more rarely after 2–3 days or only after 2–3 weeks. The pulmonary findings become manifest only after 2–3 days, occasionally only at the time of crisis and remain evident for a few days after defervescence. In every case of belated defervescence, empyema which forms in about 5% of the cases should be one of the things taken into consideration—the prognosis is usually favourable.

Treatment is that of bronchitis in a more energetic manner. The room should be warm and equable in temperature. The child has got to fight for what air it can get to its lungs, so do not allow relatives and others to crowd into the room and use up what air there is. Use the bonchitis kettle. If the weather is not too hot, a jacket poultice, hot Turpentine Stupe or Antiphlogistine should be applied to the chest; but particular care must be taken not to scald the child's skin and not to allow him to catch a chill while the heat is being applied or removed. In the early stage give Ammonia Carbonate gr. $^1/_2$, Vinum Ipecac ℥ iii, Golden Syrup ℥ viii and

water two drams, one every three hours to child one year old. If the child gets blue about the lips and face, two or three leeches should be applied over the lower part of the breast-bone. After that benefit may be obtained from Tincture Digitalis, 1 minim every two hours in a little water, in cases of exhaustion. The advent of Sulpha group of medicines like M.B. 693 or Sulphadiazine, and anti-biotics like Penicillin, Aureomycin etc., in adequate doses, begun as early as possible, have not only proved of immense advantage but have revolutionized the field of therapeutics and turned the table and reduced mortality to practically 10 per cent or less. In private practice one has still to fight the superstition, the ignorance and poverty of the masses, and cases are brought in a hopeless condition, but these newer remedies have to a very large extent taken the terror out of the disease.

The diet must consist of milk only or brandy, 10 minims every four hours for a child a year old or under and double that dose for ages upto five years, should be given.

Oxygen in these cases is a life-saving measure and it is worth sending for it at once. The gas should be allowed to bubble through the warm water at rates between 60–120 bubbles a minute and catheter should be put into the child's noses for about $1^1/_2$ inches and kept attached to the child's cheek.

Laboratory Studies of Cerebrospinal Fluid in Meningitis and Poliomyelitis

Joffe and Wells attempt to determine the diagnostic value of certain common laboratory procedures. They state that a total cell count of over 2,000 per cubic millimeter is almost certainly caused by one of the bacteria other than the tubercle bacillus. Rarely, these other bacteria may give rise to a meningitis in the early stages of which the total count is less than 100. A differential count with more than 15 per cent lymphocytes greatly indicates inflammations due to the tubercle bacillus or the poliomyelitis virus. In these two groups seldom is there fewer lymphocytes, but the acute suppurative meningitides cases occasionally do have more than 15 per cent lymphocytes. In the latter the total count would, in the great majority of cases, be over 2,000 per cubic millimeter.

Quantitative protein determinations may be of some value in differentiating poliomyelitis from the other two causes of meningitis but must be interpreted with care in cases of meningitis in the early stage. The low chloride content of spinal fluid is of distinct differential value in distinguishing tuberculous meningitis from poliomyelitis. A level below 600 is almost certainly the result of the former. Tuberculous meningitis can be differentiated with fair certainty from poliomyelitis by the occurrence of an almost uniformly low level of spinal fluid sugars in the former as compared with a level above 50 mg. per 100 cc. in the latter.—Harold H. Joffe and Arthur H. Wells, *Minnesota Med.*, 32 : 608 (June) 1949.—*American Journal of Diseases of Children.*

A CLINICAL STUDY OF THE COMPLICATIONS OF COMMON COLD IN CHILDHOOD AND ADULT LIFE

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IN this article I propose to study the complications of common cold in childhood and to compare them with the complications occurring in adult persons. My humble investigation was carried out on 80 children and 80 adult persons from October 1949 to September 1950.

Before entering into the discussion of the subject proper, let us review the present knowledge about common cold, the other name of which is coryza. Coryza is the commonest of all diseases inflicted on mankind above the narrow limits of geographical distribution and sexual affinity but has a little more affection for children; moreover, it has more liking for change of seasons. A little more affection for little humanity is dangerous because it inflicts morbidity, hampers growth and development. Common cold is characterised clinically by upper respiratory catarrh caused by a filter passing virus which is again followed by a group of secondary invaders. "Secondary infection may be by organisms already present as normal inhabitants on the inflamed mucosa *e. g.*, pneumococcus, streptococcus viridians, or micrococcus catarrhalis; or pathogenic bacteria generally conveyed by droplet, *e.g.*, hæmophilus influenzæ, streptococcus hæmolyticus, staphylococcus pyogenes" (Price). The virus, which is an air-borne one, enters through the mucous membrane of nose or of pharynx, or of both. So clinically coryza has three types of onset, (1) Nasal; (2) pharyngeal; and (3) naso-pharyngeal.

Early symptoms of each type are:

(1) *Nasal type*:—Stuffiness of nose, sneezing—at first unaccompanied by any discharge, later on discharge.

(2) *Pharyngeal type*:—Soreness of throat, unproductive cough.

(3) *Naso-pharyngeal type*:—Combinations of both complications of coryza are due to secondary organisms but are never due to virus. Usually in other viral diseases *e.g.*, small-pox, measles, a life-long immunity develops after an attack of the disease but in coryza one attack predisposes to another instead of immunity. This is the most characteristic feature of the virus responsible for common cold. Lastly, coryza has got no specific treatment.

My humble investigation about the complication of coryza was carried out on 80 children and 80 adults between October 1949 and September 1950. These cases came under my treatment not for common cold itself but for various troubles varying from headache to diarrhœa, which developed in association with common cold. In

this investigation only those cases of upper respiratory catarrh detected to have an allergic diathesis, to develop pertussis, measles etc., infectious diseases were not included, otherwise no other selection was done. Investigation revealed the following facts:

TABLE I.

1. Age.—(*a*) Children—80.

Age	Number	Percentage	Age	Number	Percentage
0—1 year	10	12½ percent	6—7 year	6	7½ percent
1—2 years	10	12½ „	7—8 „	7	8¾ „
2—3 „	8	10 „	8—9 „	3	3¾ „
3—4 „	7	8¾ „	9—10 „	3	3¾ „
4—5 „	8	10 „	10—11 „	5	6¼ „
5—6 „	9	11¼ „	11—12 „	4	5 „

(*b*) Adults—80.

Age	Number	Percentage	Age	Number	Percentage
20—25	20	25 percent	30—35	18	22½ percent
25—30	19	23¾ „	35—40	23	28¾ „

Table No. I shows that the highest incidence of complication occurs in the first two years of life in childhood, and in adult cases between 35 to 40 years. In the first two years of life growth takes place in a tremendous velocity; moreover, as we know that an infection has a depressing effect on growth, so we should be careful to prevent the complications.

2. Sex.

	Children—80			Adults—80	
Males	41	51%	Males	40	50%
Females	39	49%	Females	40	50%

In this series the number of male children is slightly higher than that of female children but it is of little significance. Complication affects both sexes almost equally in both phases of life.

TABLE II.

3. Complications.

Name of the complication	Children (80)		Adults (80)	
	Number	Percentage	Number	Percentage
1. Acute otitis media ...	17	21¼ per cent	2	2½ per cent
2. Acute exacerbation of chronic otitis media.	20	25 „	5	6¼ „
3. Acute laryngitis ...	35	43¾ „	23	28¾ „
4. Acute frontal sinusitis ...	6	7½ „	17	21¼ „
5. Acute bronchitis ...	11	13¾ „	9	11¼ „

TABLE II—*(Contd.)*

Name of the complication	Children (80)		Adults (80)	
	Number	Percentage	Number	Percentage
6. Acute exacerbation of chronic tonsillitis (septic type).	7	8¾ per cent	5	6¼ per cent
7. Broncho-pneumonia ...	7	8¾ „	3	3¾ „
8. Lobar-pneumonia ...	10	12½ „	6	7½ „
9. Acute mastoiditis ...	2	2½ „	x	x
10. Subacute nasopharyngitis ...	9	11¼ „	10	12½ „
11. Subacute pharyngitis ...	15	18¾ „	12	15 „
12. Acute gastritis ..	4	5 „	x	x
13. Parenteral diarrhœa ...	8	10 „	x	x
14. Acute pyelonephritis ...	4	5 „	x	x
15. Acute nephritis ...	1	1¼ „	x	x
16. Acute exacerbation of chr. malaria	8	10 „	8	10 „

TABLE III.

Showing the relation between age and nature of complication in childhood.

Name	Age											
	0-1 yr.	1-2 yr.	2-3 yr.	3-4 yr.	4-5 yr.	5-6 yr.	6-7 yr.	7-8 yr.	8-9 yr.	9-10 yr.	10-11 yr.	11-12 yr.
1. Acute otitis media ...	4	2	2	2	1	1	1	2	x	1	1	x
2. Acute exacerbation of chronic otitis media.	2	3	3	2	3	2	1	1	1	1	x	1
3. Acute laryngitis ...	6	4	5	3	4	2	3	2	2	1	2	1
4. Acute frontal sinusitis ...	x	x	x	x	x	x	x	x	x	1	3	2
5. Acute bronchitis ...	3	2	1	2	x	1	x	1	x	1	x	x
6. Acute exacerbation of chronic tonsillitis (septic type).	x	x	1	x	1	1	x	1	1	x	1	1
7. Broncho-pneumonia ...	3	2	x	1	x	x	1	x	x	x	x	x
8. Lobar-pneumonia ...	1	2	1	1	1	1	1	x	x	1	x	1
9. Acute mastoiditis ...	x	x	1	1	x	x	x	x	x	x	x	x
10. Subacute pharyngitis ...	2	2	1	2	2	1	1	1	1	x	1	1
11. Subacute nasopharyngitis.	2	2	1	1	1	x	1	x	1	x	x	x
12. Acute gastritis ...	2	1	1	x	x	x	x	x	x	x	x	x
13. Parenteral diarrhœa ...	3	2	1	1	1	x	x	x	x	x	x	x
14. Acute pyelo-nephritis ...	x	1	1	x	1	x	x	1	x	x	x	x
15. Acute nephritis ...	x	x	x	x	x	x	1	x	x	x	x	x
16. Acute exacerbation of chronic malaria.	x	x	x	x	1	2	1	1	1	1	x	1

Children were found to suffer from one or more complications. When the rate of incidence of each type of complication in both childhood and adult life is compared with each other, the incidence is found to be higher in childhood. The following are the common complications occurring in childhood : acute laryngitis, acute otitis media, acute exacerbation of chronic otitis media, acute bronchitis, lobar pneumonia, subacute naso-pharyngitis. Common complications of adult life are acute laryngitis, acute frontal sinusitis, subacute pharyngitis and subacute naso-pharyngitis. Some of the complications, which occur in childhood, are not found in adult cases. They are acute gastritis, parenteral diarrhœa and acute pyelonephritis.

In uncomplicated cases of coryza in childhood there is rise of temperature varying from 100° to 102°F., the younger the child is the greater is the febrile reaction. Febrile reaction is highest in the following complications, *e.g.*, acute mastoiditis, lobar pneumonia, acute pyelo-nephritis and acute exacerbation of chronic malaria. Some special features of complications will be discussed now. Acute otitis media, though it is a common complication of coryza in childhood, may pass unnoticed due to its relative painless nature. When pain occurs in the ear, the infant may cry and scratch its ear. For diagnosis of acute otitis media in the early stage, otoscopy should be done as a routine method in all cases of coryza ; moreover, it should not be forgotten that the cry of the baby during the otoscopy produces the congestion of tympanic membrane which leads to false diagnosis very often.

Acute frontal sinusitis is more common amongst adult patients. It occurs in late childhood but not in large numbers. Absence of frontal sinusitis in infancy and early childhood is due to the fact that the frontal sinus is not developed properly upto the age of six years. Percussion of frontal sinus reveals tenderness, which differentiates it from the other pathological conditions producing supra-orbital headache. Moreover, acute frontal sinusitis is usually found in patients having the deviation of nasal septum. It is useless to wait for the development of classical signs in lobar pneumonia. Early diagnosis of lobar pneumonia is essential.

Early diagnosis of lobar pneumonia depends on : *(a)* Sudden rise in the range of temperature ; *(b)* hurried respiration ; *(c)* alteration of pulse and respiration ratio which is normally 4 : 1 ; *(d)* fine rales and diminished breath sound which may be present usually in the axillary region. The lung lesion is present on the side where breath sound is feeble. Broncho-pneumonia, which is a disease with minimum lung signs, is the worst of all complications as regards its prognosis. Debility is constantly associated with it, all the three premature babies of this series developed broncho-pneumonia and later on succumbed to it.

The spasm of the larynx is found with moderate or severe types of acute laryngitis in childhood in a variable degree whereas the adult cases are free from it. In acute exacerbation of chronic tonsillitis (septic type) the severity of acute flaring up is difficult to judge from the tonsillar condition. The degree of severity should be judged on the basis of range of pyrexia and the degree of tenderness of the enlarged cervical lymph glands. The younger the child is, the greater is the severity of the affection.

Vomiting indicates the presence of acute gastritis but it is present in acute gastritis as well as whooping cough. Presence of vomiting independent of attacks of cough suggests the presence of gastritis; moreover, vomited material in gastritis contains mucous.

Both acute gastritis and parenteral diarrhœa occur exclusively in infancy and early childhood which are the result of swallowed muco-pus leading to catarrh of stomach as well as intestine due to the fact that the skill of expectoration takes the first five/six years of life to be developed.

So the treatment of naso-pharyngeal condition is of prime importance in the treatment of both acute gastritis and parenteral diarrhœa. Subacute naso-pharyngitis is usually associated with maxillary sinusitis.

As regards the acute pyelonephritis the following signs, which are given little importance in usual text books, are found, *e.g.*, *(a)* renal tenderness on bimanual palpation; and *(b)* presence of tenderness in the renal angle by Murphy's kidney punch method.

Summary.—The highest incidence of complication in common cold is found in the first two years of early life whereas in adult cases between the age of 35 to 40 years. Sex has no influence on the incidence of complication; complications of coryza occur more in childhood and are of greater severity in terms of adult cases. Some of the complications like acute gastritis, parenteral diarrhœa, acute pyelonephritis do not occur in adult life. Common complications in order of frequency occurring in childhood are acute laryngitis, acute exacerbation of chronic otitis media, acute otitis media, subacute pharyngitis, acute bronchitis, lobar pneumonia; complications occurring in order of frequency in adult patients are acute laryngitis, acute frontal sinusitis, subacute pharyngitis. Bronchopneumonia which affects usually the debilitated and premature children, ends in fatality.

Sulfonamide Dosage in Early Infancy

Sulfonamides are excreted through the kidneys in a manner similar to urea. Urea clearances are abnormally low in premature, newborn and young infants. Sulfonamides are not excreted rapidly in these infants, and, as a result, satisfactory therapeutic blood levels can be maintained with infrequent doses. The authors prefer the subcutaneous to the oral method of administration because it is more efficient.—J. B. Richmond, H. Kravitz and W. Segar., *J Paediat.* 36: 539, (May) 1950. —*American Journal of Diseases of Children.*

TREATMENT OF SMALLPOX

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Introduction.—The incidence of smallpox in large numbers in India, in spite of the specific preventive measures, calls for its prompt treatment. While most countries in the West have been able to eradicate smallpox completely, India somehow or other has been unfortunate in this respect, in spite of the same protective measures. Whatever might be the causes that have given rise to such a situation, the problem warrants a careful study. It is estimated that there are about 300,000 smallpox cases with an appallingly high death rate in India. Hence the treatment of smallpox on proper lines should occupy as much an important place as the preventive measures, any way, amongst us.

The principles of the treatment of diseases of virus origin[1], to which group smallpox also belongs, are entirely different from those of bacterial diseases. While the causative bacterium, being extra cellular, is vulnerable to chemotherapeutic agents, the virus is least vulnerable, because of the intracellular position it enjoys.

To understand the rationale of the treatment of virus diseases it is necessary to summarise the recent knowledge regarding the virus infection. Regarding the pathology of a virus disease, it may be stated that a virus, after entering the host, may come into immediate contact with susceptible cells, as in the air-borne respiratory diseases like influenza, or there may follow a period of invasion which may vary from a few days to several months, during which time the virus travels to selective tissues by way of the gastro-intestinal tract, the circulation, the lymphatics or even the neurons. During this phase it may be possible to neutralise, inactivate or even block these extracellular viruses from entering into the cells, by means of immunological, biological or chemotherapeutic agents. But viruses, once they get into the cells, seem to be particularly invulnerable to the action of either the anti-bodies or the chemical agents. However, recent researches point out that certain chemotherapeutic agents may pass through the cell membrane and inhibit the virus either directly or by their action on the host cell. The action on the host cell might involve the enzymatic processes of the infected cell as to make it an unfavourable medium for the growth of the virus. This fact probably explains as to why the induction of passive immunity through administration of gamma globulins is beneficial in cases of mumps and measles, whilst it is least effective in other diseases like influenza or viral pneumonia. There is still another group of virus diseases wherein the infectious agent, after multiplying within a cell for sometime, destroys the cell, and, after bursting out, spreads out into fresh group of susceptible cells. Smallpox belongs to this category of virus disease and I shall

be dealing about this aspect of the question in detail presently. I must emphasise here that tremendous progress has been made with regard to the treatment of these virus diseases, thanks to the newer type of anti-biotics, Aureomycin, Chloromycetin and Terramycin. Some of these anti-biotics exert specific action on the rickettsia group and the larger type of viruses like psittacosis. We have no specific chemotherapeutic agent for the '*Vaccinia-variola*' group of viruses as yet. It is quite possible that very soon we will have drugs acting specifically on these viruses also, but as it stands now none of the available chemotherapeutic agents exert any specific action. What, then, is the remedy for smallpox ? We know that drugs may act in two ways—one, specially on the causative agent as stated previously, and the other, by neutralising the toxins of the infectious agent. If there should be no drug specifically acting on the infectious agent, the next best alternative would be to aim at a drug possessing the neutralising properties of the toxins. It has been established that in very many instances, the signs and symptoms of an infectious disease are more due to the toxic factors than to the infectious agent. There has been growing evidence that this is so in most virus diseases. Herein lies the key to the treatment of smallpox. If by such a treatment (though you may not call it an absolute specific treatment) you are able to reduce the mortality rate, reduce toxæmia, minimise eye complications and prevent pitting and scarring, one has to think that it is a step forward in therapeutics.

In order to understand the principles of the treatment of smallpox, maybe, it is advantageous to recollect briefly the disposition of the smallpox virus in a smallpox patient[2]. (*Vide Chart page 658*).

When the smallpox virus enters the body, there is a temporary viræmia which may last for a very short time. Then the virus enters into the cells of the reticuloendothelial system or the lymph nodes, to grow and multiply there. In other words, the virus is intracellular during the 12 days of incubation period of the disease. The onset of the acute symptoms of smallpox synchronises with the bursting of the cell and throwing of the virus particles into the blood stream causing a secondary viræmia and toxæmia. This viræmia lasts till the viruses again get into cells—this time into their selective cells resulting in the onset of the rash. Hence the free circulation of the virus in the blood is only on two occasions—once when the infection takes place and again during the first 3 days of the disease proper—when the virus can be directly attacked by the chemotherapeutic agents. Any drug, to exhibit its specific action, has to be exhibited when the virus is freely circulating in the blood.

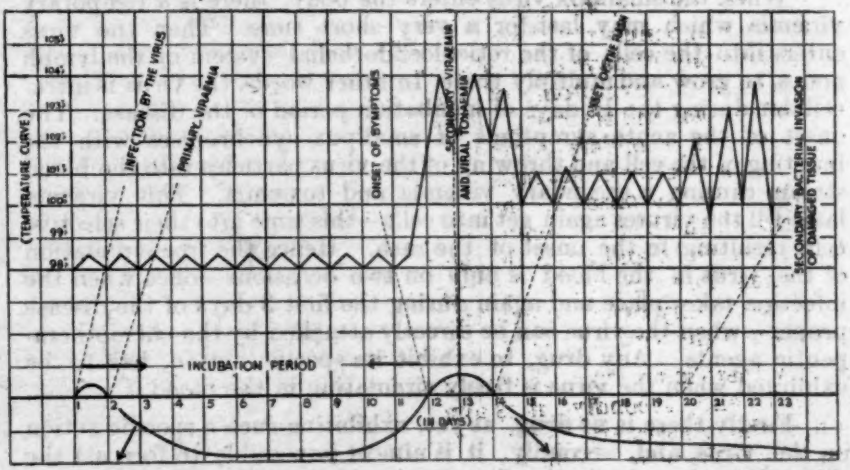
Firstly there is no drug, as yet, exhibiting such a specific action on the virus, and, secondly, it is almost impossible to forecast the time when the viruses are free in circulation. Hence the therapy by direct attack seems almost impossible. Then there is the

possibility of certain drugs interfering with the metabolism of the infected cell. Even here we have no effective drug as yet. The next alternative is the administration of drugs capable of neutralising the toxæmia of smallpox. There is growing evidence that there are certain toxic factors in the 'Variola-Vaccinia' group of viruses and these toxic factors could be neutralised by Penicillin or a factor associated with Penicillin.

Experimental work on vaccinated animals [3 & 4] at the Government Vaccine Institute, Bangalore, gave indication that combined Penicillin and Streptomycin therapy might be useful in the treatment of smallpox—Streptomycin and Penicillin acting on secondary organisms and Penicillin alone on the toxic factors of Vaccinia-Variola group of viruses. Mere spraying of the combination of these drugs was found to give the desired results. The preliminary communication (personal) from the Medical Officer, Isolation Hospital, Bangalore City, indicated that toxæmia even in very severe cases was lessened, eye complications were minimised and practically there was no pitting and scarring even in confluent cases. The scheme of treatment was extended by other Medical Officers and in one series of 16 cases, 4 were treated as control, 2 were treated with Penicillin spraying only, 2 with Streptomycin only, and 8 with combined spraying of Penicillin and Streptomycin. It was observed that all the 4 controls, one of the two cases treated with Penicillin and both the cases treated with Streptomycin died, but

CHART

Diagram showing correlation of temperature with the disposition of the Smallpox virus in the Patient.



Virus multiplying in the cells of lymph nodes and the reticulo-endothelial system.

Virus localised in cells of skin and mucous membrane.

7 out of 8 cases treated with combined spraying of Penicillin and Streptomycin survived with very little pitting or scarring. This is a clear indication that there is a definite lowering of mortality rate, complications and sequelæ. It has not been possible to correlate results with reference to hæmorrhagic type of smallpox, as cases will die long before you suspect that it is one of smallpox. In the absence of any specific treatment this scheme of treatment should be considered a definite step in advance, in smallpox-therapy.

Scheme of Treatment

1. *During pre-eruptive stage* :—It is not possible to diagnose a case to be one of smallpox during the pre-eruptive stage, but could be suspected with a certain amount of certainty in epidemic areas since the pre-eruptive symptoms are characteristic. During the epidemic season, pyrexia with backache, headache and vomiting in a person who is not vaccinated or vaccinated long back could be safely suspected as a smallpox case. The treatment in the pre-eruptive stage consists of intramuscular injection of 8000 units of Penicillin and 4 mg. of Streptomycin per kilo of body weight, split up into 3 or 4 convenient doses per day till the onset of the eruptive stage. In other words, this works out to about 5 lakhs units of Penicillin and 0·5 gm. of Streptomycin for the whole day for an adult weighing about 120 lbs. Children will get proportionately less. Once the eruptions appear the injection is replaced by the spraying described under 2. You will be required to employ this treatment rather rarely, as most cases come up during the post-eruptive stage for treatment.

2. *During post-eruptive stage* :—Combined spraying of Penicillin-Streptomycin solution is recommended in the following strength—2 lakhs units of Penicillin and 1 gm. of Streptomycin or Dihydrostreptomycin are dissolved in 200 c.c. of 25% sterile glycerine solution (three parts of sterile distilled water and one part of sterile glycerine forms 25% glycerine solution).

The solution may be painted or sprayed over the eruptions twice a day and allowed to dry. Spraying or painting is continued till the majority of scabs fall off from the body.

If the case is of a very severe type, injection of Penicillin and Streptomycin, in the dosage suggested under No. (1), may have to be employed for a few days in addition to spraying.

In addition to the specific treatment mentioned above the scheme of treatment should include the usual supportive and palliative measures. These measures are so well known and need no elaboration here.

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WHOOPING COUGH

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IT is a specific disease specially incidental to childhood. Its mortality is highest next to measles, in all the specific diseases in childhood under five years of age.

Incubation period:—Varies from four to twenty days, depending upon the atmospheric and individual conditions. It does not usually affect a person twice, one attack conferring immunity more or less.

There are two stages. The first stage, the catarrhal stage, lasts for a week or ten days; symptoms being moderate fever and hoarse dry cough. Child is poorly with short fits of cough and loss of appetite but may appear well during the day. At this stage auscultation shows moist and dry bronchial rales. As the disease progresses, cough becomes more noisy and paroxysmal with nocturnal exacerbations, face looks puffy, eyes watery and dusky. Sometimes extravasations of blood beneath the conjunctiva. Whoop appears at the end of the second week or later. Paroxysms may be short or long according to the severity of the disease, terminating with flatulent eructations or vomiting. Vomit contains mucus and food but may be mixed with a little blood.

Second stage:—The spasmodic stage varies in intensity and duration. Child may get from twenty to thirty paroxysms in twenty four hours. The characteristic sound is produced by the influx of air through parts which are not prepared to allow it to pass.

Duration of the disease is from six to eight weeks.

Age:—Children from 1 year to 10 years, but usually from two to six years of age are prone to get the disease but infant under six weeks also can be afflicted. Mortality increases when this occurs under one year.

Results and sequelae:—Emaciation, atelectasis, emphysema, caseous bronchial glands, tuberculosis and nephritis may result.

Cause of death:—Broncho-pneumonia, convulsions, wasting, atelectasis of lungs.

It is a fertile source of caseous disease of bronchial glands and tuberculosis of lungs.

Complications:—Epistaxis, haemoptysis, ulceration of frenum linguæ, convulsions, pleurisy, pericarditis, laryngitis and broncho-pneumonia out of which the last and convulsions are most important.

AETIOLOGY:—This disease occurs in epidemics specially in winter and spring season, sometimes sporadic cases do occur. It is

contagious, the contact may be actually from patient or may be with articles of clothing. The germ is a small bacillus, gram negative resembling Ba-influenza.

Quarantine period for contacts is fifteen days and for the patients it is six weeks from the commencement of the whoop.

Diagnosis is difficult in the early stage but later the whoop and ulcers on the frenum linguæ are characteristic features. Blood shows signs of leucocyotosis, there being increase in lymphocytes.

Prognosis is bad in the very young but in uncomplicated cases mortality is not very large. About one third of the cases get complications.

TREATMENT.—Differs in the two stages.

In the first stage we have to arrest the whoop. Pertussin vaccines mixed (*i.e.*, catarrhalis, pneumococci) should be given every 3rd or 5th day. Other medicines used are as follows: One minim of Glycerine of Carbolic Acid every 3–4 hours, Cresoline vapour, Resorcin 1% swabs, nasal insufflation of $^2/_3$ grains of Benzoin or Boric Acid powder once or twice at night, Paraffin oil in syrup and water. By the last remedy cough becomes less frequent and less violent. To control the paroxysm Phenazone (5 grs. three times a day to a child of 7 years), can be tried. Compound Tincture of Camphor, Potash Nitrate are also valuable drugs.

Second stage.—Tr. or Ext. of Belladonna or Quinine in large doses, Creosote ½ to 1 m. in Castor Oil Emulsion, mixture of Sodii Bicarb and Tr. Belladonna, Adrenalin 1 to 3 mg. of 1 in 1000 solution in distilled water with Glycerine, Ammon. Bromide, Potassium Bromide, Chloral Hydras, Antipyrin, Citrophen or Heroine Hcl are important drugs used in this stage.

Diet should be fluid mostly, nutritious, and should be given directly after whoop. Here I would like to write about an interesting case of whooping cough which had febrile relapse and bronchopneumonia as complications and which was successfully dealt with by Aureomycin. A child, aged four years, who had just recovered from measles was seen in the first stage. There was history of his having come in contact with a child suffering from whooping cough. The child was given an expectorant mixture along with prophylactic Pertussin mixed Vaccine, every 3rd day. About five injections were given but the disease progressed, the whoop developed, paroxysms about twenty times a day. He used to vomit about 8–10 times during day and night. At this stage curative Pertussin Vaccine was started along with a mixture containing Potassium Iodide, Tincture Belladonna, Tr. Lobelia, Vin. Ipecac, and Liquor Arsenic. He was also given extraction of Thymi Sæchat of Tæschner Co and local painting of the throat by 1% solution of Resorcin. Patient had much relief in the lessening of the intensity and frequency of the paroxysm but still had them and also the vomits persisted. This had

covered a period of about 2 months since he had measles. There was slight wasting as well, in spite of the precautions taken about diet. The child was never in bed and there was every possibility of his having exposure.

When he was apparently getting well, he had a sudden attack of fever. He was given Cibazol tablets 1/3 every four hours without any relief. Temperature persisted, lungs showing a few rales on auscultation. As the temperature rose very high at night Inj. Quinine Bihydrochlor grs. v was given (blood was taken for M.P. and was found negative next morning). Temperature receded a little and rose again to 105°F. Signs of bronchopneumonia set in, child was very much restless. Injection Penicillin G Sodium Crystalline 250,000 units was given twice a day, in all 1,000,000 being given without any change in the patient's condition. At this stage, after consulting the Civil Surgeon, Aureomycin was started, one capsule a day, ¼ capsule every four hours. With one capsule there were signs of improvement, after 2 capsules were given temperature touched normal but rose again. After two more capsules were given the patient was completely relieved of the fever and cough.

There was much wasting after this attack of bronchopneumonia and even after the recovery the child kept very low health. He was given nutritious diet together with Shark liver oil, Iron and injection Colloidal Calcium with Vitamin D. With six of these injections, the child started gaining weight.

An Epidemic of Diarrhoea of the Newborn

In March 1946, there was a sudden outbreak of diarrhœa among the babies of the obstetrical department of the Andre-Boursier Hospital in Bordeaux affecting 22 infants. Although a large number of cases of otitis media (an epidemic of 30 cases from January to April) occurred at that time, Chastrusse observed many cases of diarrhœa in babies not showing any sign of ear infection. Between the fourth and thirteenth day frequent liquid yellow odourless stools were expelled forcefully with much gas. Most patients were not very sick, but some had a toxic appearance—they had some focus of infection (usually the middle ear and antrum), became rapidly dehydrated, vomited and showed moderate temperature elevation. Under correct treatment the condition of some improved in a few days, but in other children in spite of the best treatment diarrhœa persisted and the child died athreptic and toxic or with a complication such as bronchopneumonia, abscess and so on.

Etiology:—Breast-fed and artificially fed infants were affected alike; the milk could therefore not be responsible. Parenteral infection, such as otitis media, was proved in only six of the 22 cases; therefore infection outside the intestinal tract could not be the cause either. Stool cultures still yielded no pathogens.—L. Chastrusse, *Paediatrie*, 36: 299 (July-Aug.) 1947.—*American Journal of Diseases of Children*.

RICKETS

(Described first by Glisson in 1650)

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Definition.—Rickets is a progressively wasting disease of childhood, general wasting, dehydration and osteo-dystrophy. It usually begins about the fourth month—the period of rapid growth—and lasts several years, with remissions and ultimate healing, behind traces of deformities produced during the period when the bones are soft and fragile.

The study of this malady is very interesting because of the remarkable advances in the knowledge due to combined efforts by the physiologist, the biochemist, the pathologist, the radiologist and the clinician.

Incidence:—Rickets being the disease of the period of active growth occurs between the fourth month and two years. Marasmic infants, because of their arrested growth, do not promptly show clear signs of the progress of the disease. The manifestations at the later period of life may be allied but are not termed as rickets but osteomalacia. In the infantile period it is worthwhile noting that the diet is very much restricted, whereas in later periods the child starts eating all the articles of food and hence rarely suffers from deficiency of vitamin D.

Climate:—Rickets is rare in climates with a high annual incidence of sunshine, whereas scarcity of anti-rachitic rays in the Northern climates leads to a greater incidence of the disease unless a compensatory amount of vitamin D is supplied in the diet. The situation in India has been underestimated, but still children of osteomalacic purdah women escape rickets due to the fact that they are allowed to use the sunshine freely until puberty.

The climate in every country differs in different seasons, so the incidence of rickets. It is prominent during the winter season and also in colder countries beyond the equatorial region, with its peak in the months of March and April. After the summer solstice the process of healing starts and the incidence during summers is very low. Further, the incidence is very low in high altitudes. It is very high in crowded and industrial cities, where the residential many-storied pigeon hole slums do not get sufficient sunlight and in the lower stories not at all. Also the air is stuffed with dust and coal particles, or cotton and jute fibres.

It is reported more in the babies of better classes who are superstitiously and fastidiously looked after so that not a ray of direct sunlight may fall on the body of the baby. They are usually given sunlight in a closed room with glass windows. They may well be spared from catching cold but they are as well deprived of the

most essential ultra-violet rays due to their absorption by the zinc content of glass.

Race :—This was supposed to have some effect due to the pigment on the skin of the Negroes and the Asiatics. It was supposed that the darker races were the most affected and therefore had the predisposition to rickets. But it was later conclusively established that the pigment on the body was an effective screen for the hard sunrays of the tropics. Incidence of rickets in the Negro babies was observed only when they were transferred to Northern countries when their naturally pigmented skin could not take the sparse ultra-violet rays so much beyond the equatorial region. Similarly, in India, we do not come across rickets in the villages or smaller towns. Because of malnutrition and infection due to uneducated-unhygienic life they might have so many more dangerous diseases but not rickets. In India rickets is purely a disease of the babies of the middle class and the aristrocracy—the former cannot afford to pay for a sunny house and the latter would not expose their valued children to the open sunshine. But the vast majority of the population of India are in a very low economic level and so in cities they cannot provide their babies with a superstitiously fit residence, and in other places they have no time to be fastidious about their children due to their preoccupations in the farms or in the fields.

Altitudes :—High altitudes are very favourable in the sense that they do not provide oblique rays for a very long period of the year. The rays do not have to pass through a greater depth of atmosphere and so there is lesser absorption of the ultra-violet rays.

Premature infants are notably predisposed, due to their rapid rate of growth during the extra-uterine life. It is remarkable that twins which are not properly developed but are full term do not develop the disease.

Intra-uterine rachitic tendency has been recognised, while rachitis tarda appearing during puberty is known and quite recently they are quite commonly seen due to practically famine conditions prevailing in many parts of the world, and particularly India. It was widely noted during the last war in Germany, Austria, Poland, France and the occupied parts of the U.S.S.R.

Thompson thinks that more than 50% of the infants of Edinburgh, London, Glasgow, Manchester etc., show unmistakable signs of rickets. The same figure probably holds true of the U.S.A. and much truer of the large cities in this country.

Probably heredity or pre-natal factors have some influence, such as the general health of the mother (the father also may have some, due to the weaker potency of the spermatozoa), her diet and the surroundings where she is living.

It can be seen from the above that rickets has been ascribed to almost every conceivable influence which is known to be

harmful to human beings. Defective hygiene, improper diet, intestinal toxins, bacterial infections, syphilis, derangement of the secretion of endocrinal glands and hereditary factors.

But with all this the greatest advance in our knowledge of rickets recently is the understanding of the precise nature of the hygienic factor, which has been particularised as sunlight rather than fresh air, cleanliness, exercise etc.

Some people have tried to classify the different types of the disease. For this some have taken age incidence as the criterion while others have found 'Organ affected' as a better basis. Whatever organ is principally affected it is part of the whole, also it is essentially a disease of childhood and that too the first two years of life, even though potentially rachitic states do occur even late after the growth has stopped. As such, all these classifications do not lead us to any clarification of the disease nor simplification of the process. Fœtal rickets, infantile rickets, cœliac rickets, liver rickets have been recognised. Of course what is called renal rickets is a definite and separate identity and has been described elsewhere in detail.

PATHOLOGY.—We know rickets for the last three hundred years (since Glisson first described it in 1650), still our knowledge has not reached the final stage. Even though rickets is a systemic disease our information is limited to the changes in bones only. The literature on the subject is inconceivably vast. Historically it is worthwhile going through the reviews by Park and Howland.

Long groping about the truth of the causation of rickets, has led to many remarkable discoveries. The names of Mallanaby, Sherman Pappenheimer, Mc.Collum, Simonds, Shipley, Park, Howland, Kramer, Hess and their co-workers have all contributed substantially.

At autopsy it is rather easy to cut with a knife down through the epiphyseal end of the bones deep into the shaft, the cut surface presenting an extraordinary contrast to the normal bone. Normally the line of ossification is perfectly sharp, even and is a narrow line; while in rickets this is a wide and irregular band of soft, grey and translucent tissue, with whitish opacities and gritty particles representing the calcified lamillæ. Islets of bluish cartilage are seen throughout. The cortex of the shaft shows a soft spongy growth. These changes are very clearly demonstrable in the lower end of the femur, upper end of the tibia, lower end of the radius, and most prominently in the costochondral joint. Occasionally they can be seen in the smaller long bones of the digits and the palm.

Another characteristic is the abundant growth of the blood vessels into the epiphyseal cartilage from the perichondrium, splitting up the cartilage in all the directions, making a network of tiny blood vessels running parallel to the plane of ossification. These are in layers and several such layers "Stages" may be seen at once. They become confluent with vessels of the marrow cavity as the

irregular region of ossification advances, and ultimately lose their identity. As they lie exuberantly in the matrix of the cartilage it loses its power of taking up the Meth. Blue, and is readily stained by Eosin. This collagenous material soon becomes converted by the osteoblastic activity into the osteoid tissue formed by the marrow vessels.

The significant feature is that (as shown by Pommer) the development of the osteoid tissue is in excess and at the same time imperfectly calcified. The normal bone has a narrow border of osteoid tissue surrounding the trabaculæ of the spongiosa, in rickets this osteoid tissue may, in extreme cases, even fill the whole of the marrow cavity. The tissue growing from the periosteum is regularly laid and may even be found in layers (Periosteal Callus), or be deposited as small osteophytes.

A section through the epiphysis of this bone shows that the cartilage bordering on the narrow capillaries is not regular in its columnar arrangement, neither is it uniform in its calcium deposition. Calcium is deposited in patches and clusters of blood vessels intervene deep into the cartilage substance, so that long tongues of cartilage seem to protrude in a most irregular way far down to the narrow cavity. In the remaining matrix are spread osteoblasts in a highly irregular thick laminæ—morphologically like bone—but not calcified. It is this tissue which, as described above, fills up the thick demarcation line of ossification. This tissue engulfs, as it were, the capillary bunches. The cartilage margins left behind are also covered by this tissue on the margins. This tissue is also prominent in the shaft of the bone, making up the superficial layers of the laminæ of cancellous bone and the periosteal exostoses. This osteoid tissue is present even in the denser bone cortex especially near the blood vessels of the perforating canals and near about the Havertian systems.

For a very long time the point was in dispute whether this calcium-free bone is to be regarded as the result of absorption of calcium from the previously normally developed calcified bone (Halisteresis) or new laying down of calcium-free bone. Strong evidence goes in favour of Halisteresis, even though much of this tissue is newly formed.

From the above microscopical picture we can easily deduce the various deformities that may occur to the bone. The most prominent are the enlargement of the epiphysis especially at the knees, ankles and wrists. The costochondral junctions are swollen and result in what is termed a pigeon-shaped chest, scoliosis or fractures in the long bones and their curvatures *e.g.*, Sabre Tibia. The skull is made square and the forehead is very prominent due to the spongy growth of the frontal and parietal bosses. Often there is a deep ulcer on the back of the head just where it rests on the pillow called specially as osteo-tabes.

The abovementioned enlargement of the epiphysis is due to the fact that ossification is very sluggish and the cartilage does not quickly get converted into less bulky ossified tissue, also to a lesser extent it is due to the excessive production of cartilage. This is evident by the tenderness on pressure found on pressing the bone-ends.

Spleen:—The spleen is enlarged and shows increase only in the reticulum fibres but no parallel increase in the connective tissue. Pulp is hyperaemic, Malpighian bodies are small and giant cells are present (Hayaski). However, distinct increase in the connective tissue is reported by Boid.

Clinically abdomen protrudes and in many cases the spleen is much enlarged and firm, though of course not so hard as the malarial cake. *Ostitis fibrosa* is in fact the same process taking place in the bone marrow but in a modified form producing a complicated picture.

It is remarkable that a return to the normal architecture of the line of ossification is possible during healing.

From the above observations of the clinician, the pathologist and the radiologist, we find that there is in every case a deficiency of calcium and phosphorus—still more their proportion is disturbed during the activity of rickets, and that vitamin D, ultra-violet rays and sunlight hold a key position in the causation of this deficiency.

No one single factor could be set to be the causative agent. Clinical observations have suggested the importance of vitamin D and the ultra-violet rays, while experiments on animals go to show that it is the adequate quantities of Calcium and Phosphorus, or more precisely the deviation from the normal of their ratio in the blood plasma, that is the real starting point.

Experiments on rats have definitely shown that the absence of vitamin D or ultra-violet rays do not necessarily cause rickets. But withdrawal of Calcium and Phosphorus along with it definitely causes rickets and particularly so if the relative proportion of this is abnormal. The unbalanced proportion of the bone-forming minerals in the diet is of much greater importance than their absolute amounts.

This Calcium and Phosphorus ratio which is too high or too low, though their actual amount respectively is not withdrawn or reduced, causes the disease, which in turn is arrested and cured by the addition of vitamin D or ultra-violet rays. This points to the fact that vitamin D is concerned with maintaining the ratio of Calcium and Phosphorus in the blood plasma more than anything else.

It has long been thought that high carbohydrate diet has anti-calcifying effects and so favours infantile rickets. Mallanaby confirmed this by his experiments with cereal such as wheat, oatmeal,

white flour, and rice. But in fact there appears to be no relationship with carbohydrates, proteins or mineral contents of these materials and their rachitogenic properties which are clearly overcome by the addition of vitamin D or irradiation of the cereals. Results of Bruce and Callow further proved that this socalled anticalcifying or rachitogenic property of the cereals is due to the unavailability of phosphorus of the cereals which is present only as Phytic Acid. This Phytic Acid easily destroyed by the addition of Hydrochloric Acid releasing Phosphorus as well as calcium and magnesium, thus favouring the calcification.

The Calcification Process

Wells, Howland, Watts and Robinson have put up four different theories of the calcification out of which that of Robinson seems to be more approximating to the facts. He holds that it is an enzymatic action by which the fluids in the immediate vicinity of the cartilage and the periosteum get supersaturated with Ca_3PO_4 and he has also demonstrated the presence of this enzyme in the blood. This enzyme is capable of hydrolising different phosphoric esters. He has named this enzyme as *Phosphatase*.

If we take the existence of this phosphatase as a fact then it stands to reason that vitamin D, ultra-violet rays or irradiation does stimulate the activity of this enzyme.

Calcium and Phosphorus of the Body

Calcium constitutes about 2% of the weight of the adult body, and about 99% of the total quantity is contained in the (bone) skeleton. Muscles contain only about 8 mg. per 100 gms. of wet weight, plasma or serum from 9–11·5 mg., R.B.C. have only minute amounts. Blood as a whole contains 4·5–6 mg., eligible Ca. is laid down during the first five months of intra-uterine life, while 60% is deposited there during the last two months of ante-natal period.

Greater part of the calcium in bones is in the form of Calcium Phosphate. Though the importance of P. ion was not recognised for a very long period the credit is due to the experimental pathologist who proved that rickets could be produced in animals fed on diet poor in phosphorus even though it had abundant calcium. And this is the main advance in our knowledge of rickets. Previously whole attention was focussed only on calcium and its behaviour in the body. In rickets the organic phosphorus content of the blood is reduced while the Ca. is unchanged, but the ratio of the ions in the bone is, even in severe conditions, not changed though the total amount of ash is diminished.

There should be Ca. 10 mg., P. 5 mg. per hundred cc. of blood. In any case this proportion must be maintained. Howl and Krammer assert that if the Ca. multiplied by P. results in a figure below 40 you are surely dealing with rickets. Vitamin D raises the

volume of calcium and phosphorus and thus maintains the ratio as well as the quantity, both to normal.

Due to the lowered blood calcium infantile tetany is a constant accompaniment of rickets and *vice versa* because of the relative disutility of the higher amount of phosphorus if present. But some experiments were carried out by Dibbelt before the influence of Vit. D. or U. V. Rays was known. He mentioned that rickets was not observed even with calcium-and phosphorus-free diet, but cod liver oil was given, naturally meaning thereby that the presence of Vit. D. surely is concerned in the necessary mobilisation of Ca. and P. from other parts of the body to be utilised for ossification of bones.

It is now generally believed that these salts are not present as a mixture in the bone but they are as a complex compound, and the ratio of Ca. to P. is 2·2 : 1 (or 10 atoms of Ca. to 6 atoms of P.) and the residual Ca. 2 : 1 (9 atoms Ca ; 6 atoms P).

Pathological calcifications at other places are believed to have the same chemical composition as that of bone.

Bone ash amounts to 60% of its dry weight. In this ash Ca. is 36%, P. 16%, Mg. 0·5%, Co_3—5·5%. The ratio of Ca. to P. is 2·2 : 1.

$Ca\,CO_3$ is 13%, $Ca_3\,(PO_4)2$—80%, Mg. 3, (PO_4) 2-2.

Rickety bones contain lesser ash and larger percentage of water and organic matter. Mg. content of bone is said to be higher in rickets.

The deficiency of calcium and phosphorus in the bone is probably due to the failure of absorption from the intestines. The blood P. figure is usually low in rickets, *i.e.*, Ca. 6 and P 2 mg. per 100 c.c. (Normal figure Ca. 10 and P 5 per 100 c.c.). As shown above the mobilisation of Ca. and P. is done by the action of phosphatase. This also occurs in the intestinal mucous membrane, the kidneys and the blood.

The plasma phosphatase is "expressed" as the No. of Mg. of P. liberated from the excess of *Beta* Glycerophosphates in 48 hours at 38°C. and at pH 7·6 by one c.c. of plasma (Hunter).

The normal figure is 0·15 mg. In rickets the plasma phosphatase is increased during the active stages of the disease showing that it is not being utilised for mineral mobilisation.

It must here be pointed out that the bone is not simply an inert supporting substance but a living tissue, whose mineral and other composition fluctuates under the influence of other body functions. Aub and his associates have shown that the trabeculæ constitute a storehouse of calcium for ready use whenever necessary, even by other tissues in case of deficient supply. This is perhaps why the blood Ca. level does not come down for a pretty long time even on a Ca. free diet.

Bone also deposits lead, radium, fluorine, arsenic and so may be said to have detoxicating effect for removing these substances from blood circulation and depositing them in the bone.

COMPOSITION OF BONE.—Osseous tissue consists of organic material (mainly protein), water and minerals. Water is 25%, organic material 30%, inorganic constituents 45%, chiefly Ca, P. up 15–18% of fresh bone Kn. Na. Cl. Fl. and Fe. Calcium makes Mg. and smaller quantities of and 20–25% of dried bone after extraction.

Calcium in blood exists in two forms, diffusible, non-diffusible. The non-diffusible form is bound to the proteins, while the diffusible form is easily dialised and is subject to ultra-filtration. It is in an ionised condition such as Calcium Carbonate or Phosphates. The non-diffusible and un-ionised form is in a very small quantity and exists as Cal. Proteonate (McLean and Hastings).

Inorganic salts like Chlorides, Carbonates, Lactates, and Gluconates are readily absorbed ; but Cal. Phosphates (less soluble) is not so easily absorbed. The absorption starts quickly after injection and takes the peak by about two hours and in an hour more the blood level is again normal. In fact it is not possible to mention the high level of blood calcium for any appreciable time.

Calcium in food is present as organic or inorganic salts. Probably it is absorbed only in an inorganic form and that too from the upper parts of the small intestine.

Sugar, especially lactose, favours absorption of calcium, due to production of organic acids in the intestines.

Fats free of vitamin D lower the Cal. absorption owing to the formation of insoluble Cal. soaps.

Protein foods tend to increase the absorption due to the formation of soluble proteonate complexes with amino acids.

Milk is the best of all the sources of Cal.; but quite large amounts (up to 0·2 gms. daily) are obtained from hard drinking water. Vegetable Cal. such as in carrots is easy for absorption, while those containing Oxalates cannot do so, due to the relative insolubility of Cal. Oxalates. The cereals (wheat, barley, oats) contain phytic acid which forms an insoluble salt of calcium and so require addition of inorganic acids to dissolve it and make the calcium available.

Cal. is excreted in the lower part of the small intestine and through urine (it is not excreted in the large intestine). Even during fasting the excretion continues, Cal.-free diet also does not affect the excretion. Thus under such conditions a negative Cal. balance is established (difference between injestion and excretion—balance). The balance is positive during growth, pregnancy, acromegaly, while in rickets this is negative.

Vitamin D

Vitamin D.—Search for anti-rachitic vitamin makes one of the most interesting stories of biological science.

The anti-rachitic vitamin (D) like the parathyroid glands (probably in combination with) affects the calcium metabolism. It is supposed to increase the solubility of Cal. and Phosphorus in the blood as also its conservation.

It is soluble in fats, oils, ether, alcohol and is insoluble in water. The absorption band is between 260–270. This anti-rachitic vitamin belongs to the class of substances known as Sterols, or Solid Alcohol such as Cholesterol, Phytosterol, Ergosterol. The vitamin has been isolated in crystalline form and named as Calciferol, symbolically as D.

As long ago as 1890 Palm suggested that sunlight had anti-rachitic properties. In 1910 Huldchinisky successfully employed ultra-violet rays from a mercury lamp and in the meanwhile Mallanaby published his results proving the anti-rachitic value of Cod liver oil, egg yolk and butter and so on. There has been a big chain of workers with intensive research in this direction.

Roseinheim, Webster and others found out that Cholesterol of animal origin, and other sterols of vegetable origin were on irradiation useful as anti-rachitic, hinting that these sterols were precursors of vitamin D. Thus a final link between vitamin D and irradiation was established. These observers later found that it was in fact the Ergosterol which was really responsible for this effect, and it was named Provitamin D.

The Ergosterol on irradiation yields Lumisterol:—Tachysterol, Calciferol appearing in order as given. Calciferol is not the end product, but excess of irradiation gets decomposed into Toxisterol which has a very bad effect on calcium metabolism and is highly toxic. Calciferol is the least toxic of the whole chain and most efficacious.

It requires to be noted that the later workers have abandoned the older conclusions that Ergosterol was the Provitamin. It has been shown finally that the vitamin D after irradiation of Ergosterol is not identical with that produced by the irradiation of skin. Waddell found that the Cholesterol achieves the same potency as the Cod liver oil and much greater than irradiated Ergosterol. He concludes that there is substance in association with Cholesterol and is activised on irradiation. This is provitamin and is quite different from Ergosterol. According to him there is no evidence of the existence of Ergosterol in the human skin in any appreciable amounts.

It has further been experimentally proved that irradiated Ergosterol is much less potent than Cod liver oil in its anti-rachitic

property. Irradiated Cholesterol is also much more potent than Ergosterol. Further on they have isolated the substance that is activised on irradiation—7-Dehydro-cholesterol. This is the real provitamin D of the mammalian skin. Waddell's findings offer an explanation for certain discrepancies which have been noted in the past. Before Waddell many observers had already concluded that there must be some difference between the vitamin D of Ergosterol and that of Cod liver oil origin.

After this new orientation the Calciferol from Ergosteral has been termed as vitamin D_2, while the activated 7-Dehydro-cholesterol is called vitamin D, (there is no vitamin D_1) and these are the only two sterols of medical interest.

Vitamin D is produced on irradiation of skin, milk, mother hens, and naturally present in Cod liver oil and this is 7-Dehydro-cholesterol. That produced on irradiation of yeast, Ergosterol etc., is vitamin D_2. Both have different structural formulæ.

Sources of vitamin D:—Halibut liver oil is the richest source of vitamin D. Cod liver oil and other oils from bony fishes also have a rich store. But Cod liver oil contains only 100 I.U. of vitamin while the Halibut liver oil contains 1200 I.U. per gram of oil.

Mammalian liver is rich in vitamin A but is very poor in vitamin D.

Bills thinks that the fish is able to synthesise this large store of vitamin D as so much quantity could not be supposed to have been acquired from food, and irradiation of the skin of these is not possible because they are deep water fishes.

Other sources are butter, milk, egg-yolk cream. The anti-rachitic property of all these depends upon the diet of the parent animal, as well as the amount of the sunshine that they get. Their anti-rachitic property is considerably reduced in the winters and is much more in summers.

Pasturisation does not destroy or even reduce the anti-rachitic property.

Vegetable foods as a rule are very poor in vitamin D. It is absent from vegetable oils unless they are irradiated. Fruits and green vegetables contain but an insignificant amount. Yeast of course has high content of Ergosterol and on irradiation acquires quite a high degree on anti-rachitic potency.

Ultra-Violet Rays

All this points to one single fact that sunlight has the most important role in the prevention of rickets. Nearer the Sun lesser is the incidence of rickets and *vice versa*. It has finally been established that it is the ultra-violet rays of the spectrum that are of any use, and still it is the rays of shorter wave length. Hess and others have demonstrated that the ultra-violet rays of 2800 Augstron

unit wave length are the most effective, and the effective range is between 2600 and 3150 A units.

Park and Elliot have proved that these effective wave lengths are filtered out by the depth of the atmosphere, dust-laden atmosphere. When the locality is at a greater distance from the Sun such as happens in winter, the rays have to traverse a greater medium and obliquely. This is the cause of the seasonal incidence in places outside the equatorial zone. In this way sunrays give the maximum supply of ultra-violet rays on the summer solstice and minimum on the winter solstice.

Dust and coal particles also absorb the short rays of this end of the spectrum, hence a child living in Bombay, Calcutta, Cawnpore, Indore, Tata Nagar and such industrial places is in greater danger of rickets than those living in thinly populated or non-industrial areas.

Hess and Lungaden have shown that the inorganic phosphorus of blood of infants is in direct proportion to the existence of these rays in the atmosphere. The average value 4·35 mgs. per 100 c.c. in June is reduced to 3·92 mgm. in December and continues like that upto March.

The shortest rays from the Sun which can reach most localities on earth have a wave length of about 290 *mu.*, while those from the carbon arc or mercury lamp are around 220 and 180 respectively.

Ordinary window glass due to its zinc content filters out all those rays having a wave length shorter than 320 *mu.*

Ultra-violet rays do not penetrate the skin more than about ½ m.m.

In general the incidence of rickets is rare in places with an abundance of sunshine.

X-RAY PICTURE.—On X-ray examination of a well developed case of rickets, the osseous deformities are clearly visible as osteoporosis. Density of the bone is less in the whole skiagram. Bone ends are not clearly defined, but have a woolly appearance—'moth eaten'. The contour of the articular ends and of the bone are concave —"cupping" and not convex as found in a normal bone. This appearance is due to the lack of, or irregular distribution of, the minerals in the zone of preparatory calcification. Normally this (line of ossification) is clearly defined, and is almost a straight band next to the unmineralised layer of proliferating cartilage.

Skiagrams of early rickets or slight attacks are not very definitely identifiable, as against the normal bones of the same age. Some physicians have definitely demonstrated the changes in infants otherwise looking normal and thus nipped the disease in the bud.

But in an established case the appearance is characteristic. These changes are best seen at the wrists, ankles, knee-joints and costochondral joints.

During healing this line of ossification gets on clearer reducing the cupping, and the metaphysis shows mineralisation and casts a deeper shadow than the epiphysis. As against this an unaffected bone gives a uniform shadow all over.

Skiagram is an aid to the diagnosis of rickets, but it must be borne in mind that the disease will have progressed a good deal before it could be clearly diagnosed by an X-ray picture.

The characteristic changes first appear in the lower end of ulna and the lower end of femur, and so early photograph should be of these parts.

Clinical Findings

In this country the patients are neither brought for routine examination nor for consultation with rickets in view. The child will usually be brought to you for gastro-intestinal disturbance of varying degree, general emaciation, tetany, ulcers round about the mouth or in the mouth, general dermatitis and less often for delayed speech, delayed dentition, laxity of the extremities and inability to stand, sit or walk properly or without support. Still less occasionally he will be brought for deformities particularly when hardening has set in and it is beyond correction. Practically always you have to go through your routine method of examination and try to detect rickets, if any. This writer has made it a point to keep rickets in mind while examining every child below seven years.

The cardinal sign is the *rickety rosary* or the bead-like swellings of the costochondral joints on both sides. Eversion of the free ends of the ribs and consequent depression above the rib border, "Harrison's Sulcus", and a peculiar conical appearance of the sternum giving general appearance of the chest like that of a pigeon. This is particularly found if the child has suffered from co-existent lung diseases such as pneumonia.

Bossing of the skull:—Swelling on the frontal and the parietal bones is very prominently visible. It gives the typical appearance of a square head, bulging brow, flat face and depressed fontanelles. Advanced cases show what is called *cranio-tabes* (thinning of parietal and occipital bones) due to constant postural pressure. Quite frequently there is ulceration on the occipital bone and a typical *egg-shell crackling* could be felt in extreme cases.

The spine shows the effects of general osteoporosis, and the infants who have begun to sit or stand show off kyphosis, scoliosis, flat foot, all being the effects of mechanical pressure, along with muscular hypotonia. Pelvis may also get an irregular shape for the same reason.

The limbs are generally lax, joints loose, bones softened, muscles flabby, and in advanced cases deformities such as Genu Valgum, Genu Varum, Coxa Varathe, most prominent being the bent tibia "Sabre-tibia". The epiphyseal ends of the limbs look enlarged and

they are tender on pressure. Gingers often show a fusiform appearance.

Relation to dental caries is not well established while it is noted that rickety children always show improper calcification of their teeth. In fact rickets is always detected during the teething period.

Earlier the rickety children are typically fat and flabby, with hypotonic muscles. Flatulent distention and pot belly due to laxity of the abnormal muscles are quite common.

Respiratory:—Constant cold, coryza, bronchitis in an infant should suggest the possibility of rickets.

Apart from these signs there is marked effect on the other activities of other organs. Deficiency or absence of one vitamin also disturbs the balance of all the other vitamins, *e.g.*, deficiency of vitamin D will cause retardation of fat absorption which in turn reduces the absorption of the fat soluble vitamin A and causes xerophthalmia. Again due to deficient utilisation of Carbohydrate other water soluble vitamins may also be in short supply. In the opinion of the writer we should always presume the deficiency of multivitamin. Of course that which is particularly short will always show off in a symptom complex due to that vitamin.

The deficiency of vitamin causes inefficiency in the functions of all the organs such as liver, spleen, pancreas, kidneys. All of them are liable to secondary infection easily because of reduced resistance.

As a rule every child must be thoroughly examined for rickets since we always get a complicated case. Differential diagnosis is not of great significance. And still we would not be justified in overlooking any point. The first among the signs of omission is the failure to examine all the infants and children that are brought to us, for rickets, even during the presence of all the other emergencies. Detection of rickets is bound to give us a great help in the treatment of any other serious complication.

COMPLICATIONS.—As a matter of fact we never come across a pure case of rickets. Invariably it is complicated with some other malady which takes up the priority and the patients are brought for that and not for the original malady. Nevertheless chief complications are mentioned hereunder:

Tetany:—This is characterised by hyper-irritability of the nervous system, leading to convulsions, laryngismus stridulous, carpopedal spasms, head spasms (Spasmus Nutans), and a peculiarly distinguishable nystagmus. The blood calcium is reduced. Park and his associates term this also as a kind of rickets where instead of phosphorus there is a deficiency of calcium.

Infections of respiratory tract:—Pneumonia, bronchitis, cold and catarrh (influenza). Of these bronchitis and catarrh are very common and are due to the reduced resistance of the body to these infections. Pneumonia of course gives a very grave prognosis.

Anaemia:—There is general weakness due to calcium deficiency and reduced fat absorption. This directly results in dehydration and reduced reticulogenic power of bone end and the flat bones. The patient of rickets is always anæmic. A form of anæmia known as "Von Jaksch's anæmia" is a constant accompaniment but rickets has not yet been proved as its cause.

Hydrocephalus:—A mild degree of hydrocephalus is always present. To some extent at least the appearance is due to enlargement of frontal bosses. Superficial cranial vessels are dilated and the fontanelle also look larger.

Gastro-intestinal disturbances:—This is a constant accompaniment, and quite often the rickets is detected while examining in a routine way. These attacks are repeated with vomiting and loose motions which turn green after a time (interestingly enough the mother attributes this colour to her diet of green vegetables and intentionally stops the diet thus reducing the injection of vitamins as well as Chlorophyl and other minerals).

DIFFERENTIAL DIAGNOSIS:—We shall only deal with the bony conditions as that would occasionally require differentiation from osteogenesis imperfecta, achondroplasia, chondro-osteodystrophy, syphilis and scurvy.

1. *Osteogenesis imperfecta*:—Multiple fractures in the ribs may simulate the beading of rickets, but they will not be in a line necessarily and there may be two or more beads in one and the same rib. The parchment condition may not be limited to the frontals and the parietals. The family history and the blue sclerotic will seal the diagnosis. In doubtful cases X-ray will clearly decide even the achondroplasia and chondro-osteo-dystrophy of Brailsford-Morquito type.

2. *Syphilis* often is difficult when the case presents ulcers in the mouth and generalised ulceration on the skin. Other stigmata of syphilis will remove the doubt and blood examination of the baby as well as the parent will leave no doubt.

3. Scurvy will be evident with multiple hæmorrhages.

PROGNOSIS.—The prognosis generally is very favourable. Majority of the cases are so mild that they pass off unnoticed and recovery starts by the fortuitous advent of spring, or accidental change in diet and locality having a lot of free sunlight. These cases leave no deformity. The cases that have stayed on for a longer period and in which mechanical pressure has brought about changes such as curved tibia, kyphosis, scoliosis, square head, flat foot, do not revert to normal shape though the lesion heals up completely. The rickets only softens the bones but the deformity is due to the mechanical or postural pressure and the normal pull of the muscles. The healing and calcification proceed side by side with the

deformity. Thus the deformity is hardened due to calcification if correcting measures are not taken during the period of healing.

In this country we see these permanent deformities very commonly, especially severe head flat foot, curved tibia and distorted pelvis. Deformity of the pelvis is particularly dangerous in girls as it is a direct and permanent obstruction to childbirth. Dwarfism with curved bones is very often seen in the streets. The buffoons of circus are usually rickety people. In this country, stunting of growth due to untreated or late treated rickets is an every day affair and all these cases may be traced to have suffered from malnutrition and avitaminosis.

TREATMENT.—Prophylaxis and treatment are practically the same but for the fact that more intensive measures are taken on the curative side.

The attack should be from all the sides at a time so as to hasten the time of healing, that is, before the deformities could be permanent, Sunlight, Irradiation, Irradiated Diet, Multivitamin by mouth or by injection may all have to be employed.

The minimal daily requirement of vitamin D for a human being has not been accurately established, since such metabolic studies are very complicated and difficult to control as compared to those in which laboratory animals can be utilised. From whatever data is available the National Research Council of U.S.A. has decided that the optimal requirement ranged between 400–800 international units daily.

In view of the above normal requirement we might assume that nearly double the amount may be required for treatment and so we keep our range for treatment from 800 to 1500 units daily, while making allowance to probable destruction in the stomach due to disturbed gastro-intestinal condition during the activity of rickets. Along with vitamin D we must also give requisite amounts of other vitamins in order to keep up their synergic action.

Selection of the product of course requires one's own experience. This is important because we are getting a lot of spurious drugs these days, and the market is flooded with them. It is a well-known fact that the Government of India examined all the Cod liver oil preparations that were available in the Indian market (local and foreign manufacture). Out of over a hundred samples tested, majority did not contain vitamin D at all while only 17 samples contained vitamin D but not to the extent mentioned on the label.

It is very regrettable that the Government did not disclose the proprietary names or their manufacturers.

In advanced cases we have to give more and immediate attention to the gastro-intestinal disorder, and unless we correct this, any administration by mouth would be all vomited or purged out undigested. Demulcents in an emulsion form are indicated. In this

drop doses of Ol Ricini would be of great use. In the meanwhile vitamin D may be given by injection and repeated until the stomach recovers its retentive power.

Dehydration, if any, has to be taken quite seriously and subcutaneous Saline may help a lot.

Oil massage to the whole body helps to tone up the muscles and activate the skin due to improved circulation.

Another serious complication that is present is bronchitis and pneumonia. Proper attention with Sulpha drugs, Penicillin, massage with Lin. Terebenthene to the chest and other necessary things may have to be given. It is worthwhile pointing out here that the Sulpha drugs in such a case be used with great caution. Firstly they have the tendency to aggravate vomiting; secondly, they cause leucopenia; thirdly, its aftereffects on the liver—we have very often traced infantile cirrhosis after Sulpha treatment for a long time. In spite of all these Sulpha drugs have their place though they have to be administered with caution.

PHOSPHATURIA.—Very often the patient is brought to you for phosphaturia, with no other visible sign of rickets. On examination of course some clue somewhere is available. Urine examination may along with phosphates show albumin. The avitaminosis has probably affected the filtration of the kidneys. Alkaline diuretics with vitamins are indicated.

Infant's Diet in Relation to Intestinal Flora

The investigators give the details of a study of the intestinal flora in all portions of the intestinal tract of 54 well and sick newborn infants with all types of feeding.

The organisms isolated were Bacillus coli in 68 percent, Lactobacillus bifidus in 44·07 percent, Enterococcus in 40·7 percent, Bacillus proteus in 25·9 percent, Staphylococcus (albus and aureus) in 18·5 percent, Streptococcus lactis aerogenes 14·4 percent, paracolon bacilli 11·1 per cent, Neisseria orbiculata 7·6 percent, Alcaligenes fecalis 5·6 percent, Clostridium perfringens 3·8 percent and Cryptococcus (Torula) 1·8 percent. The authors also made a similar study on 50 white rats.

The authors concluded, briefly, that feeding is responsible for little difference between the intestinal flora. Lactobacillus was found in both sick and well infants receiving all types of feeding. They never succeeded in isolating this organism in aerobic conditions. In the rats and mice L. acidophilus, which is aerobic, predominated over the anaerobic lactobacillus bifidus. The authors were not able to prove the predominance of one organism over another in different feeding, although it would appear that L. bifidus is more abundant when the quantity of lactose in the milk product is greater.

The flora of children suffering from nutritional diseases of a nonspecific nature is similar in most respects to that of the normal child. After the sterile meconium has passed, the intestinal flora of the faeces in the newborn is almost identical with that of the nursing baby of 6 months of age.

Except in the stomach and the first part of the small intestine, there is no definite relation between the type of flora and the anatomical portion of the digestive tract.—F. Gabrero Gomez, J. Monereo Gonzalez and B. Taracena del Pinta, *Acta pediat. espan.*, 87: 209 (March) 1950.—*American Journal of Diseases of Children.*

SOME OBSERVATIONS ON EPIPHYSEAL INJURIES IN CHILDREN

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AMONG the injuries of bones in children the epiphyseal injuries are likely to be missed often but these require early and efficient treatment. The epiphyseal injuries are more often treated as sprains and the nature of the lesion is realized only when the disorders of epiphyseal growth occur.

Those who want to treat injuries in children must have a very good knowledge of the various epiphyses, their variations, numbers, dates of appearance, fusion etc. Otherwise many mistakes are likely to be made, such as mistaking an unusual epiphyseal line or double epiphysis with a line separating them as a fracture line. A comparison with the contralateral side is a useful guide.

ETIOLOGY.—Usually indirect violence is the cause of epiphyseal injuries. Epiphyseal injuries may happen as an isolated lesion or as an avulsion fracture separation in association with dislocation; for example, in outward dislocation of the elbow, the internal epicondyle epiphysis is avulsed and displaced outwards and downwards. As in other injuries, boys sustain epiphyseal injuries more than girls.

SITES.—Any epiphysis may be injured. The most common one involved is the lower radial epiphysis. The others usually injured in the upper limb are the upper radial epiphysis, the capitellar epiphysis of the humerus, the internal epicondyle epiphysis of the humerus, the olecranon epiphysis of the ulna or various combinations of these, such as the upper radial and ulnar epiphysis or the ulnar epiphysis and the medial epicondyle or the external condyle epiphysis of the humerus. In the lower limb, the lower tibial epiphysis is the one involved oftener; the upper tibial or lower femoral epiphysis may be involved; the femoral capital epiphysis is likely to slip and its pathology etc. are somewhat different and are beyond the scope of this paper.

Nature of the lesion.—The writer has noted three types of epiphyseal lesions, *viz.*:

(1) The so-called "typical epiphyseal lesions" *i.e.*, the lesion includes the whole of the epiphysis, the epiphyseal cartilage and a triangular bit of the metaphysis. (*Figures 1 and 2, vide page 680*).

(2) The "vertical compression fractures" resulting from compression or buckling on the vertical axis; the epiphysis is crushed, the amount of crush depending upon the degree of violence. (*Figures 3-a and 3-b, vide page 680*).

FIG. 1.

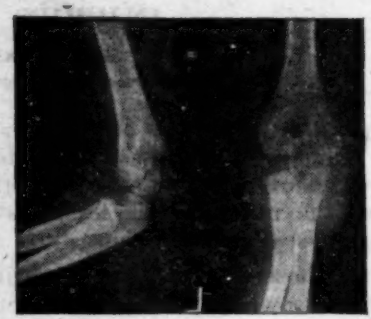

FIG. 2.

FIG. 1.—A case of the so-called typical epiphyseal separation of the lower radial epiphysis with backward and outward displacement and fracture of the ulnar shaft.

FIG. 2.—A case of external condyle epiphysis of humerus with minimal displacement.

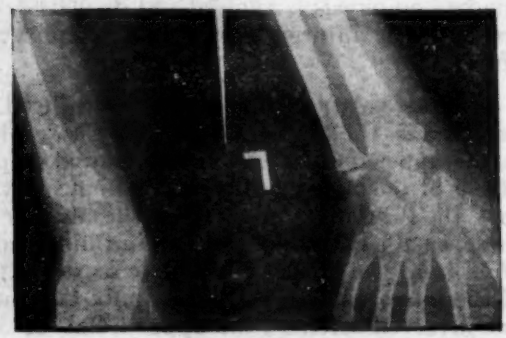

FIG. 3 (a)

FIGS. 3 (a) and 3 (b).

A case of bilateral vertical compression fracture of the lower radial epiphyses; the right one [Fig. 3 (b)] is associated with a backward displacement and fracture of the ulnar shaft.

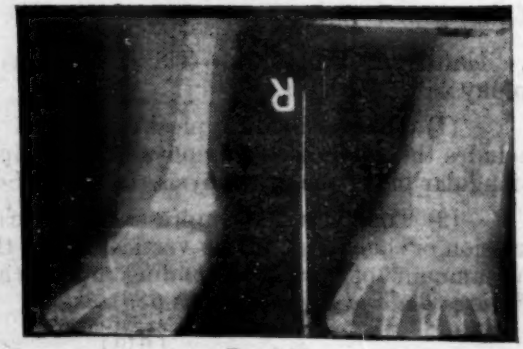

FIG. 3 (b)

(3) Ephipyseal separation through the epiphyseal plate of cartilage itself; this is an occasional one. The treatment and prognosis depend upon the type of the lesion.

SIGNS AND SYMPTOMS.—There is usually a history of fall from a height, such as a fall from a tree, or into a well, or from the roof of a house, followed by pain and a varying amount of swelling in the region of an epiphysis. In lesions with marked displacement, deformity is evident. In type (1) cases without displacement, or in type (2) cases there may not be any deformity and the swelling may be only minimal. The localized tenderness is always present. Hence, after a fall, when there is a complaint of pain and localized tenderness in the region of an ephiphysis, a radiograph is taken to confirm the diagnosis and to know the type of the lesion. If there is any doubt, the normal side is also radiographed and compared.

DIAGNOSIS is easy if the history, symptoms and signs are carefully noted and a radiograph is taken. The writer wishes to impress one fact in examining the radiographs. The writer has noted in many cases of injuries to the bones of forearms and legs after landing on the palms of feet as the case may be, in addition to the usual types of green stick and complete fractures, vertical compression or buckle fractures also. In these vertical compression fractures in the forearm and the leg, if one bone is involved, the other is also examined for the same lesion; if the lesion is not seen at the same level in the other bone, the one or the other of its epiphysis is examined for vertical compression injury of the epiphysis (*Fig. 4*).

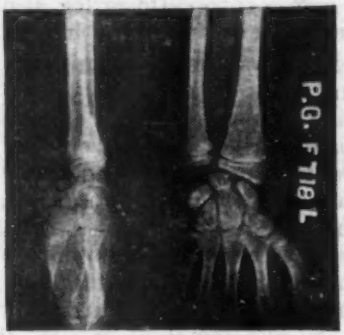

FIG. 4.—A case of buckle fracture of lower third of left radius; there is injury to the lower ulnar epiphysis also.

PROGNOSIS regarding the future growth of the epiphysis depends upon the type of the lesion and the promptness with which it is attended to. In type one lesions, if treatment is done promptly and efficiently, the prognosis is good. In types 2 and 3, we cannot predict exactly the amount and nature of the epiphyseal damage. There is usually a varying amount of disordered growth and premature fusion. Hence we have to be very guarded in giving the prognosis in these cases regarding the future bone growth.

Complications and sequelæ.—1. *Disordered epiphyseal growth*:—There is a varying amount of disordered growth either

uniformly or irregularly. Irregular growth leads to deformities (*Figures 5 and 6*).

FIG. 5.—A case of old dislocation of elbow with fracture separation of internal condyle. Note the disproportionate growth of the lower end of the humerus with varus deformity.

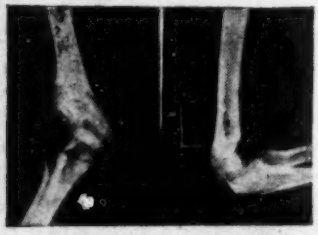

FIG. 6.—A case of old unreduced fracture separation with displacement and rotation of external condyle epiphysis. Note the small size of the external condyle epiphysis and the disproportionate growth of the lower end of humerus.

2. *Premature fusions*:—In the case of forearm or leg, if one of the epiphyses undergoes premature fusion and the other bone overgrows the companion bone and leads to deformity, *e.g.*, if the lower radial epiphysis fuses earlier, the lower end of the ulna overgrows and deviates the wrist outwards (valgus) and sub-luxates the joint. In the case of the lower limb not only the deformity is developing in the same limb but due to premature fusion there is also discrepancy in limb length (*Fig. 7*).

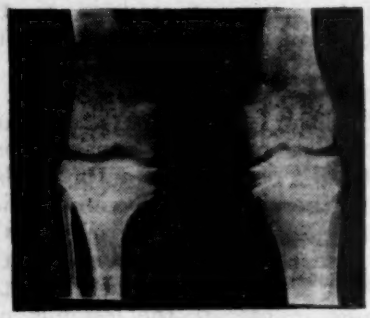

FIG. 7.—A case of old injury to the upper tibial epiphysis resulting in premature fusion. There is a shortening of two inches in the limb length as compared with the normal. Note the fibular upper epiphysis is still growing (compare with the normal side).

3. (*a*) *Nerve lesions*:—As a result of the trauma, nerves may be injured; *e.g.*, ulnar nerve is injured in medial humeral epicondyle epiphysis separations.

(*b*) Tardy paralysis of nerves due to callus or commonly due to increasing deformities, *e.g.*, untreated external humeral condyle epiphysis leads to increasing valgus deformity and tardy paralysis of the ulnar nerve.

TREATMENT depends upon the type of the lesion. In type (1) and (3) cases, if there is no displacement, the limb is immobilized for a duration depending upon the site, usually in the upper limb four weeks and in the lower limb eight to ten weeks.

If there is displacement, under general or intravenous anæsthesia, the fragments are reduced into position. The success of reduction depends upon the promptness with which it is attended to. Reduction within a few hours of injury is easy and perfect. If delayed, reduction is difficult and often impossible. Some of the displaced fragments require open reduction and fixation *e.g.*, external condylar fracture separation of humerus with gross displacement and rotation of the fragments both in the horizontal and vertical axis (*Fig. 8*), reduction fails more often and open reduction and fixation of the fragment by soft tissue repair is necessary. So also in green stick fractures of the neck of radius (*Fig. 9*), if the closed reduction fails, open reduction without opening into the periosteum is done and the results are good.

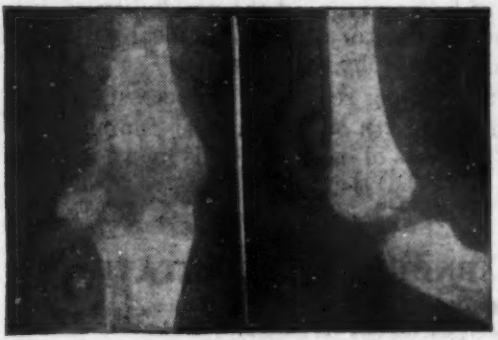

FIG. 8.—A case of fracture separation of external condyle epiphysis with gross displacement and rotation in a vertical axis; closed reduction failed, necessitating open reduction.

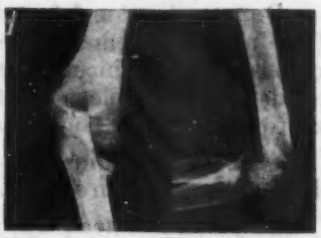

FIG. 9.—A case of green-stick fracture of neck of radius tilted outwards. Reduction was possible by closed manipulation.

In type (2) cases, the compression can be released by traction but nothing can be done for the damage already sustained by the epiphysis. We have to watch the epiphysis for disordered growth or premature fusion. If this happens, we have to prevent the development of deformities in the case of the upper limb and in the case of the lower limb we have, in addition, to attend on the discrepancies in the limb growth. In irregular growth of the epiphysis-producing deformities, epiphyseodesis (by open operation, the entire epiphyseal cartilage is destroyed) is done or better still the growth is controlled by the Blount's method of "epiphyseal stapling." In the case of the forearm, if the epiphysis of one bone undergoes premature fusion, the growth of the corresponding epiphysis of the companion bone is controlled by epiphyseodesis or better by stapling. If one lower limb is not growing as the normal one, control of the growth of the normal limb is considered.

Summary.—I. Three types of epiphyseal injuries are noted and the recognition of the type is helpful in giving the prognosis and treatment.

2. The importance of having in mind the possibility of injury to the epiphysis when there is a history of fall and localised pain and tenderness in the region of the epiphysis is stressed.

3. Early diagnosis and proper reduction is necessary.

4. In case of irregular growth of an epiphysis, epiphyseodesis is advisable. Treatment in cases of early fusion is mentioned.

TWINS WITH ABNORMAL PRESENTATION

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ONE day in the early morning I was called to attend a case for delivery in a village 1½ miles away from my place. As it was an urgent call I went there at once with my equipments.

The patient was a multipara having no child alive. She was twenty-eight years old. To my surprise she was in a bad condition since twenty-four hours and two *dais* were watching her.

On examination I found that there was abnormal enlargement of the uterus, œdema on feet, and bladder distended high as she had not passed urine since twenty-fours. Lungs N.A.D. (nothing abnormal detected). Spleen and liver were normal. Heart beat increased due to prolonged labour pains. Pulse was rapid, *i.e.*, 115 per minute.

On P. V. examination I felt head with bulging membranes which are not ruptured still.

On palpation head was fixed in the pelvic brim. I found out fœtal heart sounds which were slightly audible. The second head was felt in the right hypochondrium and second fœtal heart sounds were not clear.

The present stage was no uterine pains due to twins and prolonged labour. That is partial uterine inertia.

As there was no other medical help possible I started my own line of treatment.

TREATMENT.—First with a septic technique I emptied the bladder. I gave full assurance to the patient and I injected Pethidine Hydrochloride (Roche) 1 c.c. to give rest and sleep.

After a short period pain started, then Pitocine (P.D. & Co.) 1 c.c. was given. With psychic courage and confidence she began her labour pains. I explained to the *dais* the presence of two babies. Then Glucose 50% 25 c.c. with Calcium 10 c.c. was given. The

membranes were ruptured and liquor-amini came out. As the cervix was fully dilated I gave her Pituitary 1 c.c. (P.D.) She started labour with strengthened pains and the first baby came out. No sooner the baby came out, the second child was seen at once with breech presentation. She was given rest for a short period and then pains started. The lower extremities were seen and pulled out by rotation. But there was a good deal of hæmorrhage. The second Pitocine 1 c.c. (P.D.) was given. The whole delivery was conducted with caution and upper extremities came out. But to my surprise the cord was round about the neck and so impacted in it. The patient was exhausted; so she was again given rest for a short period. The third Pitocine was given. But in spite of all my efforts she did not deliver the child. The first placenta had also not come out and forces to bleed. So, lastly, I rotated but failed to do so, then I put a finger in the mouth and applied jaw flection and shoulder traction method and slowly pulled downwards and forwards with traction on the shoulders. The cord was separated at once and both the placentas were re moved bi-manually to save the woman from undue bleeding and ensure safety to the babies. The first one was male and the second was female with separate cord and placenta to both.

After completing these the patient was given the following line of treatment:

(1) Anti-tetanus serum 1 c.c. I.M.

(2) Inj. Neo-gynergen (Sandoz) 1 c.c. I.M.

(3) Inj. Procaine Penicillin (Aqueous) 4 lks. (Merck's) I.M.

(4) Inj. Glucose 50% 25 c.c. with Coramine I.V.

(5) Mist. stimulant oz. i t.d.s. Mist. Ergot with Quinine oz. i, t.d.s.

(6) Sulphadiazine 2 tablets t.d.s. for 7 days.

Then for a week I visited and continued to give Penicillin Procaine 4 tablets, and Inj. Neo-gynergen 1 c.c. with above mixtures and tablets.

Conclusion.—(1) She had not developed any complication such as shock, septicæmia, postpartum hæmorrhage.

(2) Twins with abnormal presentation and cord round about the neck with impaction.

(3) The mother was saved with two babies without any undue injury in a rural area.

Present Trends in Penicillin Therapy

This is an up-to-date discussion on penicillin therapy, and emphasis is placed on the fact that penicillin is used promiscuously, widely and frequently without indication. Bacteriologic study and diagnosis are lacking more frequently than they are found. Reactions to penicillin, usually allergic, may occur. The development of penicillin procaine during recent months has been a far-reaching step in prolonging the activity of the drug. The inhibition of renal excretion may be of value in the prolongation of penicillin activity.—Sam Boyer Jr., *Minnesota Med.* 31: 1305 (Dec.) 1948.—*American Journal of Diseases of Children.*

COMMON DIGESTIVE DISORDERS OF INFANCY

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THIS simple subject is particularly selected, because the writer is of opinion that if these diseases are approached with more exhaustive investigations and with deeper views and deeper interest from the beginning itself, infantile mortality can be reduced to a great extent.

At the outset, these diseases appear to be very simple but if they are not diagnosed correctly and tackled properly from the beginning itself and if they are neglected thinking that they are only minor ailments, they may lead to grave prognosis or devitalising sequelæ. Hence the selection of this subject to infuse more stimulus to the medical and health authorities.

Only the commoner ailments which we come across in daily practice will be dealt with, leaving out the un-important ones. Further, as it is simply meant for the busy practitioner, the subject has been approached only from the clinical standpoint, with little reference to problems of ætiology and pathology but giving more weight to the treatment. Now coming to the subject proper, the chief cause of these digestive disorders, is due to the neglect of the principles of feeding. The most common disorders are :—

I. Indigestion.
II. Vomiting.
III. Colic and flatulence.
IV. Diarrhœa.
V. Constipation.

I. Indigestion.—This is a condition where a particular person's digestive system is unable to digest and assimilate the particular quality and particular quantity of food taken in. The chief causes of it are overfeeding, unsuitable food, mismanagement in feeding and chronic naso-pharyngeal infections. It usually occurs soon after the period of weaning or after debilitating diseases. Chiefly there are three varieties of indigestion, namely—

1. Protein indigestion.
2. Fat indigestion.
3. Carbohydrate indigestion.

1. *Protein indigestion* :—This is usually met with in infants who are fed with cow's milk, the caseinogen which is converted into large tough curds giving rise to mucous catarrh of the stomach and intestine.

The most prominent symptoms are vomiting of curds and mucous with colic and screaming. There may be some loss of weight. The stools are alkaline and contain a few green lathery-looking curds.

TREATMENT.—In severe cases, it may be necessary to remove the casein entirely from the diet. But more often it is sufficient to dilute the milk or to substitute a dried milk or whey. But

sometimes it may be necessary to modify the casein by citration, acidification or peptonisation.

2. *Fat indigestion*:—Fat indigestion is particularly likely to occur in young infants who are brought up from birth on full cream brand of dried milk. At first, the fat is excreted in the form of soaps and the stools are bulky-formed and pale in colour. Urine smells the odour of ammonia. Buttocks get reddened and even ulcerated. Later, free fatty acids are passed in the stools which become loose, strongly acid and offensive, causing much pain and screaming. Vomiting is not prominent but considerable weight is lost.

TREATMENT.—Initial dose of castor oil should be given to clear the bowels. The amount of fat should be cut down. In milder cases, the full cream dried milk is to be replaced with half cream milk, or if cow's milk is being used, the fat should be skimmed off. In more severe cases completely skimmed dried milk such as special Cow and Gate's milk or Horlicks malted milk has to be used for a week or two and then half cream brand to be started. In the case of poor patients, butter milk may be used as it contains less than one percent of fat. The excoriated buttocks have to be treated with some soothing application as Zinc Oxide with Cod liver oil or Milkan ointment with Cod liver oil.

3. *Carbohydrate indigestion*:—It occurs in infants who are fed with badly balanced foods having an excess of sugar or proprietary foods containing too much of starch. The symptoms arise from excessive bacterial fermentation in the intestine. Distention of the abdomen, due to accumulation of gas, causes a ventral bulge between the tightened rectimuscles, when the infant sits up. Attacks of colic, with screaming and drawing up of legs, are frequent and an excess of flatus is passed. The stools become loose, frothy and green. They are acid but not so offensive as that of fat indigestion. Vomiting is not an early symptom. In the beginning they put on weight due to the amount of water retained in the tissues, but they are flabby. In course of time, the gain in weight becomes arrested and severe degree of wasting may eventually occur.

TREATMENT.—Bowels must be cleared up with an initial dose of castor oil. The amount of carbohydrate in the diet should be cut down and starchy supplements such as barley water should be entirely withheld. A simple mixture of milk and water will improve this variety of indigestion to a great extent. The milk should be sweetened with a mixture of Dextrins and Maltose since this is less fermentable than cane sugar, glucose or lactose. About a teaspoonful of this mixture, in the form of Dextri-maltose or Daltose, added to 4 ounces of milk, brings the carbohydrate content up to about 6 percent. Protein-milk, which is acidified milk is also very useful and convenient. Prolonged feeding with low sugar, may lead to constipation and in such a case, the amount of sugar should

be gradually increased. If there is diarrhœa also, the following mixtures will be useful:

℞	Chloral Hydras	... gr. iii
	Gum Acacia	... gr. xx
	Ol. Ricini	... ♏ xv
	Aqua menth pip. ad	... ʒ iii, 1/3 part thrice a day.
	or	
℞	Soda Bicarb	... gr. v
	Tr. Nux Vomica	... ♏ ii
	Spt. Ammonia Aromat	... ♏ ii
	Inf. Gentian Co. ad	... ʒ iii, 1/3 part thrice a day.

II. Vomiting.—Vomiting is an act in which food swallowed through the gullet is ejected out involuntarily. It is not a disease by itself, rather it is the symptom of some other disease, chiefly indigestion.

For the sake of convenience of description it may be divided into four varieties namely: (1) Acute vomiting. (2) Chronic vomiting. (3) Cyclic vomiting. (4) Rumination.

1. *Acute vomiting*:—This is usually the result of an attack of gastric catarrh or indigestion. Usually there will be slight fever and occasionally high fever. The tongue is thickly furred and breath smells sour. The vomiting which occurs at the beginning of the specific fevers and intracranial diseases should be differentiated. If the child is only few days old and if the vomiting is projectile and accompanied by constipation some congenital obstruction such as stenosis of the œsophagus, pyloric obstruction or imperforate anus, should be thought of. Worms also cause vomiting very often. Vomiting of indigestion occurs about half an hour after food and it is more common in bottle-fed infants.

TREATMENT.—The most important part of the treatment is starvation. Though it is said that infants bear starvation badly, they withstand the complete withdrawal of all nourishment for two or three or even more days without any disadvantage, provided they are kept warm and enough of water is being administered.

Gastric lavage *i.e.*, washing out the stomach, will also be of great help. It is so safe and pleasant to the infant that none should hesitate to employ it. The barrel of a glass syringe is fitted to a rubber tube of two feet length, and this is in turn connected to a rubber œsophageal tube of the size of a pencil, through the intervention of a small piece of glass tube. About 10–12 inches of it is quickly passed over the tongue into the œsophagus. The funnel is filled with a solution of Bicarbonate of Soda (a teaspoonful to a pint) and about 3–4 ounces of the solution is allowed to enter the stomach. Now the funnel is inverted below the level of the child and the gastric contents including pieces of curds and mucous are siphoned off. This is repeated until the washings are clear. This should be employed daily for the first two or three days and, then, on alternate days.

Calomel 1/10 to 1/6 grain every half an hour for six doses and followed by Bismuth Carb. in 5–10 grain doses may do some good. Many times, Cerium Oxalate in 1–2 grain doses every fourth hour will be very effective. If there is dehydration treatment mentioned for cyclic vomiting should be started.

2. *Chronic vomiting* :—This is purely the result of some form of digestive disorder. There exists some sort of chronic inflammation of the stomach. This may be accompanied by progressive loss of weight, in which case it should be considered seriously. The loss of weight is due to want of nourishment to the body. Vomiting also occurs due to chronic inflammation of the duodenum which partially obstructs the passage of food through it.

TREATMENT.—The habit of rocking the child just after food may be the cause of vomiting. In such cases, any movement of the child after food should be condemned and this alone may be sufficient to stop vomiting. In other cases, regulation of the diet may be sufficient to set right this complaint. Vomiting, if it is habitual, means that the child is getting too strong a mixture of milk. Hence it should be diluted further, or a more digestible form should be substituted. The time of feeding should be regulated, taking care that feeds are not given too often. Strict rule must be enforced to feed the baby by the clock, *i.e.*, once in 2½ or 3 hours only. No food should be given in between. In these cases of vomitings too, lavage of the stomach is useful. In many cases, Bismuth will relieve the complaint if it is given in large doses as below :

℞ Bismuth Carb	grains	xii to xxx
Cerii Oxalas	grains	iii to x
Fl. Pulvis		iv

One powder four times a day in honey or milk.

In some cases the following mixture may be of advantage.

℞ Dilute Hydrochloric Acid	♏ iii
Glycerine	♏ x
Aqua Menth Pip	ʒ iv

One dram four times a day. In some cases Tincture of Iodine half to one minim in one dram of water given every hour may relieve the condition dramatically.

3. *Cyclic vomiting* :—This is characterised by attacks of severe vomiting associated with severe prostration and heavy ketosis, the attacks reccurring at regular intervals. The first attack usually appears before the second birth-day and there is a natural tendency to disappear as adolescence approaches. It usually occurs once in three weeks to three months. It is more prevalent in better classes,

probably because they consume more of fatty foods, cream and emulsions.

The attack may be sudden, but usually there will be premonitory symptoms of irritability, drowsiness, anorexia and tainted breath for two or three days. The incessant vomiting starts, even water being returned. Tongue becomes dry and lips parched. There is thirst, headache, moderate pyrexia, abdominal pain, scanty urine with albumin and ketone bodies. As the attack progresses drowsiness passes into coma and eyes become sunken and the condition becomes alarming. As a rule the condition abates after three or four days, fatal issue being rare.

TREATMENT.—Gastric lavage helps to retain the food in the stomach for longer periods. Glucose and Soda Bircarb are necessary to combat the ketosis. If these cannot be given by mouth due to excessive vomiting, 10% Glucose with Soda Bicarb (dram to a pint) should be given rectally by drip method. If dehydration is very severe subcutaneous (2½ percent) or intravenous (10 percent) Glucose in Normal Saline may be given. To avoid further attacks, a diet rich in carbohydrate and low in fat should be continued. Skimmed or half-cream dried milk suits well.

4. *Rumination*:—This is nothing but regurgitation of milk from the stomach into the mouth where it is chewed and tasted and the greater part is swallowed again. But a portion of it is spilt out of the mouth. This is repeated again and again and the loss of nourishment may be considerable enough to cause loss of weight. This is found mostly in bottle-fed infants. When once the trick is learnt, it quickly becomes a habit which may be difficult to eradicate.

TREATMENT.—Supervision by a trained nurse, use of thickened milk with groats or arrowroot and use of a linen bonnet to the head with tight side-straps to limit the movement of the lower jaw, are the procedures worth trying.

III. Colic and flatulence.—Usually these two conditions exist side by side, one having some inter-relation with the other. Colic is the painful contraction of the intestines and is chiefly due to irregular and too frequent feeding and flatulence is the condition where gas is present in the stomach and intestines causing discomfort.

The causes of colic are: (*a*) acidity due to fermentation of milk, producing lactic and other acids which irritate the bowel and induce over-contractions in its wall; (*b*) existence of undigested casein or curds in the intestine; and (*c*) the presence of gas in the bowel.

The chief symptom is pain, expressed in the shape of persistent screaming. The abdomen is hard and tender. Little coils of

firmly contracted intestine standing out, may be felt. As the child screams his legs are drawn up to flex the thighs. The screaming may be so extreme that the child may pass into convulsion. The screaming is temporarily discontinued after the passage of flatus.

TREATMENT.—During the attack, application of warmth to the abdomen by friction with warm oil or by poultices, to relieve the suffering to some extent, will be beneficial. Warmth may be applied internally by means of warm enemata, which relieves the pain by expelling gas and fæcal matter. Chilling of the abdomen should be prevented.

The following carminative mixture may be very useful for the expulsion of the flatus:

Soda Bicarb	...	gr. viii
Soda Citras	...	gr. v
Tr. Zingiberis	...	℥ x
Tr. Card Co	...	℥ x
Spt. Ammonia Arom.	...	℥ x
Spl. Aetheris Nitrosi	...	℥ xv
Aqua Menth pip.	...	℥ i

One to two drams every 4th hour. In the case of older infants one minim of Tr. Belladonna per year of life may be added to the above mixture per dose.

For the prevention of further attacks, feeding must be strictly regulated, feeds being given once in $2^1/_2$ or 3 hours depending upon age. As the swallowing of warm milk relieves the colic for a short time, the mother will be tempted to feed the child more often which worsens the pain and a vicious circle is set up. If the casein in the milk is too much for the child's digestion a few teaspoonfuls of lime water or two grains of Soda Citras before each feed will prevent future colic. Constipation should be attended to. If it still persists weaning of the child should be considered. In the case of bottle-fed infants want of cleanliness in preparing milk is the chief cause of colic. If the content of casein is very abnormal, it must be reduced further. In obstinate cases, opium in the form of Codein 1/40 to 1/30 grain may be given in a dram of Glycerine once or twice a day. Or Dover's powder 1/8 to 1/4 grain may be given especially if there is diarrhœa also.

IV. Diarrhœa.—Diarrhœa is a condition in which fluid stools are passed several times a day due to the abnormally rapid passage of the food residue through the alimentary canal. This is more common in bottle-fed infants. The disease appears to be an unimportant one and as a consequence there is a tendency to neglect it in the early stages, as a result of which the prognosis may become grave. In our country out of 22,000 deaths due to diarrhœa 17,000 were infant babies of less than one year of age. Hence this disease requires more attention on the part of the medical and

health authorities. Education of the public to create health conscience, especially regarding imperfect feeding and proper preventive measures, may help a great deal to produce this mortality.

Chiefly diarrhœa may be divided into four groups namely: 1. Dietetic diarrhœa. 2. Infective diarrhœa. 3. Parenteral diarrhœa. 4. Lienteric diarrhœa.

1. *Dietetic diarrhœa*:—Errors in diet is the chief cause of this variety of diarrhœa. The fault may be in various directions such as excess in quantity or excess in fat content or carbohydrate content of diet. Especially rich people are over-anxious about the growth of their children and hence they lavishly administer fatty oils, emulsions and sugars. This variety of diarrhœa is distributed evenly throughout the year and as a rule the illness will be mild in contrast to the infection variety.

2. *Infective diarrhœa*:—This term implies that the diarrhœa is due to an infection. Cultures show streptococci, B. proteins, B. enteritis sporogenous, B. lactis aerogenes, B. paratyphosus, dysentery bacillus, Morgan's bacillus and Sonne bacillus. It is possible that chemical changes consequent upon the bacteriological decomposition of milk also play a part. The incidence of this diarrhœa varies in different seasons, being highest in the late summer months.

Hence the synonym summer diarrhœa. It is also called acute gastro-intestinal infection, acute ilio-colitis or cholera infantum. In recent years this variety of diarrhœa is reduced to a great extent due to encouragement of breast feeding, improved hygienic methods in the collection and distribution of milk, enforcement of boiling or pasteuring of milk and increased use of dried milk. But it can be further reduced by enforcing better hygienic and medical facilities especially to the poor.

3. *Parenteral diarrhœa*:—This term is used for those cases in which diarrhœa is secondary to conditions outside the gastro-intestinal tract, such as acute otitis media, mastoid disease, acute pyelitis, meningococcal meningitis, acute respiratory infections, and eruption of tooth. This is often acute in onset and unless the existence of this variety is borne in mind it may be easily overlooked. This may be avoided by thorough examination of throat, ear, urine etc., in all cases. The diarrhœa, of course, has to be treated, but unless the primary disease is also dealt with, the treatment is likely to prove ineffective.

4. *Lienteric diarrhœa*:—This is a condition in which an urgent desire to defæcate occurs with slightly colicky pain during meals or immediately afterwards and may make the child rise hurriedly from his meal to pass motion. It may accompany chronic indigestion. But usually it occurs in nervous children and it is due to an unduly sensitive gastro-colic reflex. Opium is the only drug of choice which can be relied upon for this variety of diarrhœa.

GENERAL SYMPTOMS.—Dietetic diarrhœa will be usually mild and is preceded by some digestive disturbances. The onset may be insidious, stools gradually increasing in numbers, or it may be acute with vomiting and profuse diarrhœa as the initial symptoms. About half a dozen stools are passed in a day which are at first green, slightly offensive and often contain fatty curds and mucous. The temperature is raised by 2 or 3 degrees, anterior fontanelle depressed and the infant is miserable and constantly wailing. Loss of water through the stools causes the weight to be reduced and restricts the output of urine. In more severe cases of infective and dietetic diarrhœas, the onset is usually brisk and there may be an initial convulsion, and vomiting is an early symptom. Diarrhœa is urgent and stools soon become watery and odourless. Now the temperature rises to 103°F or 104°F. and the child becomes pale and restless with sunken eyes and wrinkled skin due to dehydration. The tongue becomes dry and red and papillæ raised. The abdomen becomes flabby and sunken and visible peristalsis of the intestines seen. The pulse becomes rapid rising to 160 or moreand heart sounds are soft with tic-tac character. Later, when toxæmia increases, the face becomes livid and pinched, respirations shallow, pulse uncountable and temperature usually falls to subnormal and restlessness changes into apathy. Death seems to steal over the child and may be heralded by one or two final convulsions. In cholera infantum, the onset is sudden with violent purging which quickly changes into rice water appearance. Toxæmic symptoms such as staring eyes, sighing respirations, rapid irregular heart and hyperpyrexia quickly supervene. The symptoms that foretell the grave prognosis in a case of severe diarrhœa, are anuria, late convulsions, scleroma and broncho-pneumonia.

PROPHYLAXIS.—Shifting the child to better hygienic surroundings especially when there are outbreaks of diarrhœa in the neighbourhood, enforcement of boiled milk, avoidance of chill and regular feeding with correctly balanced milk may help a great deal in preventing the outbreak of diarrhœa.

TREATMENT.—The three principles that underlie the treatment especially in severe cases are:—(*a*) To clear the bowel of any food or toxic material; (*b*) to replace the last fluid; and (*c*) to rest the bowels for a day or two. Unless the child is in a state of collapse, an initial dose of castor oil (half to one teaspoonful for every 6 months of age) should be administered to clear the bowel, or ½ to 1 grain of Calomel may be given. Gastric lavage is also very useful, especially so, if vomiting also is present. Lost fluid should be replaced by giving sips of water, 5% Glucose water or Albumin water (one ounce per hour). If this is not possible due to severe vomiting Normal Saline with 5% Glucose should be given rectally by drip method, after washing the bowel. If this also fails, it should be given subcutaneouly, intravenously or intraperitoneally.

Plasma or blood transfusions also may be considered. In severe cases the child should not get anything other than warm water or glucose water for 24 to 48 hours. Then milk without fat i.e., skimmed milk, can be given for a week or two. If the patient is too poor, whey with egg water or buttermilk may be substituted instead. Later arrowroot may be added in the case of older children. If the stools are green 5 minims of castor oil may be given thrice a day, for its constipating effect. If the stools are acid and the vomiting is severe, Bismuth Carb in large doses may be given thrice a day, as below :—

(After the bowels are completely cleared).

℞	Kaolin	... gr. xxx
	Bismuth Carb	... gr. xxx
	Soda Bicarb	... gr. vi
	Pulvis Creta Aromaticus	... gr. x
	Mucilage	... ♏ xxx
	Aqua chl$_3$ add	... ℥ vi, one to two drams thrice a day.

Opium in the form of Dover's powder 1/8 grain or Tincture Opii 1/8 minim for every 3 months of age, is a most valuable drug when diarrhœa is severe in proportion to vomiting and stools are green and offensive and in cases of ilio-colitis. But great harm would be done and even deaths may occur if administered when the tongue is dry and furred and the patient is in a state of collapse. Neither should it be given in the first stages before elimination of toxins. The over-dosage is easily avoided by omitting the drug when the patient is drowsy.

For cholera infantum Mistura Pro-diarrhœa 5 to 15 minims every half an hour will be very useful. When the motions are many and watery, whatever the cause may be, "Hordenol" (manufactured by Laboratories G. Beytout, Paris) $^1/_2$ to 2 c.c. be given subcutaneously daily, often with dramatic results. If the patient is too young the same preparation may be administered orally $^1/_2$ to 2 c.c. four times a day. In the case of infective variety especially due to dysentery and Sonne bacillus chemotherapy with Succinyl Sulphathiazole, Formo-cibazol or Sulphaguanidine will be very effective. When the stools are profuse and contain lot of mucous and appear like those of bacillary dysentery (and even in cases of bacillary dysentery itself) Hydrargyri Perchloride in minute doses often gives wonderful results. One drop of Liquor Hydrargyri Perchloride is dissolved in one pint of water and a teaspoonful is given every half an hour upto 4 or 6 doses. The next day 3 such doses may be repeated.

Instead of this solution two globules of Mercurous Corrossivus, 300x may be used per dose. The same effect is obtained by administering 1/32 grain of Perchloride of Mercury thrice a day, to the nursing mother. In many cases this drug works dramatically,

and, if it is effective, cure will be obtained within 24 hours. Hence it is worth trying in obstinate cases.

When the stools are watery and offensive, astringents such as Tannigen or Tannalbin or Silver Nitrate, will be useful. Silver Nitrate 1/8 to 1/6 grain may be conveniently given in 5 minims of Glycerine. The same effect is obtained by giving a decoction prepared from the rind of pomegranates.

When there are signs of collapse, stimulants like mustard-bath or brandy or spirits Aetheris Nitrosi in doses of 5–30 minims may tide over the danger. When stronger stimulant is indicated 1/4 minim of Liquor Strychnine B.P. upto 6 months of age and 1/2 minim for older infants or Camphor-in-oil 1/4 to 1 cc. should be given hypodermically to tide over the situation.

Constipation.—It is a condition in which the passage of fæces, through the intestines is delayed due to defective diet or due to defective secretion of the intestines.

The most common cause of constipation is underfeeding as a result of which very little residue reaches the lower bowel enough to be passed only every second or third day *i.e.*, there is a lack of stimulus. If insufficient amount of water is given, though the stools are larger, they are hard and dry, so much so that they are delayed in expelling. A high ratio of protein at first tends to produce large, hard constipated stools, while a low proportion of sugar has the same effect. Congenital stricture of anus or upper part of the rectum may be another cause of constipation. It is a prominent feature, in hypertrophic pyloric stenosis and Hirschsprung's disease (idiopathic dilatation of the colon). It is also a common feature of mental defect, such as cretinism. Constipation is largely a matter of habit, the children being either incapable of appreciating the ordinary demands of Nature or too lazy to respond to them. Congenital atony and impaired innervation of the colon due to chronic inflammation of the intestinal mucous membrane, as in intestinal tuberculosis, may also give rise to constipation. Chronic obstruction of the intestines such as new growths, worms, sterocoliths, or bands or adhesions or redundant colon also may lead to it. The chronic habit of administering large doses of castor oil twice daily from birth especially in the Andhra Province, also must be a cause of chronic constipation by producing a sort of chronic inflammation leading to atony and defective intestinal secretion.

Symptoms.—When the stools are dry and hard the infants are likely to be restless and they will be screaming before defæcation. There will be visible peristalsis of the intestines and colon in severe and chronic cases. In some cases of constipation the infant may be passing motion daily but the bowels are not emptied completely. In some cases there may be vague symptoms like furred tongue, unpleasant breath, cold and blueness of the extremities, peevishness, anorexia, headache, lassitude, flatulence and

abdominal discomfort. In some cases there may be evening rise of temperature for months due to auto-intoxication which may be cured with more aperients. Convulsion is a common accompaniment of constipation of children especially below three years. Due to repeated straining, umbilical hernia, prolapse of the rectum and rarely inguinal hernia and piles also may occur. Wasting is another result of constipation. In spasmodic constipation there is colicky pain due to spasmodic contractions of the bowel, which creates an obstacle to the onward passage of fæces. There is another group of constipation which is due to defective secretion of intestine, pancreas or liver and in these cases the stools are exceedingly hard, friable and chalk-like and these hard pellets may have to be scooped out of the rectum, as even strong purgatives may not be able to expel them out for days together and the rectum may be seen bulging out with these hard pellets and the child screaming and restless. This sort of constipation is seen in many cases of infantile biliary cirrhosis.

TREATMENT.—The importance of training the baby to open the bowels at definite hours of the day, cannot be exaggerated. For, many cases of resistant constipation owe their origin to lack of such training. If you go on disobeying the reflex to answer the calls of Nature, after some time, the reflex becomes dull and in course of time it becomes almost dead. After each meal, the baby should be made to lie down, over the extended legs of the mother for some time, and should be taken away after the bladder and bowels are emptied. By such assiduous effort, the infant should be made to learn the good habit of passing the fæces and urine, only at such particular hours daily. So when such habits are wanting, our object should be to educate the bowel to move spontaneously. We should not go on administering drastic purgatives, but we should use only laxatives or mild aperients which exert a more or less tonic action on the bowels. Massage of the abdomen, kneeding the contents in the direction of the colon will do good, by increasing the tone. In mild cases, laxatives like fruit juices must be tried first. One of the most harmless, effective, easily available, palatable and nutritious laxatives, is the decoction of dried grapes. About 4–6 tolas of dried grapes are to be boiled in 4 ounces of water reducing it to two ounces and the decanted fluid is administered to the baby once or twice a day. In majority of the cases, this will be quite sufficient to set right the bowel, provided it is continuously used for a pretty long period. In addition, orange juice or syrup of figs also may be given. In the case of older children 'Papaya fruits' which contain plenty of papain will be very efficacious, if given daily, especially if liver is affected. Where the motions are hard, plenty of water given between the feeds would relieve the condition. In other cases, Liquid Paraffin may be tried. If the food is deficient in carbohydrates, addition of cane sugar or brown *demerara* sugar may correct the constipation. Cane sugar is

much better than glucose or ordinary sugar. If the infant is breast-fed, addition of fat and meat to the mother's diet may be useful. But the practice of administering purgatives to the mother (to relieve the constipation of the infant) should not be encouraged. If the infant is bottle-fed few drops of salad oil or few grains of manna may be added to the child's feed once or twice a day. It is very difficult to treat constipation in a child who is accustomed to the daily use of large doses of castor oil from birth. In such a case, the tone of the intestine is lost. Castor oil has an initial purgative effect followed by constipation and this requires further castor oil to move the bowel the next day and a vicious circle is formed and gradually the daily dose of castor oil has to be increased and the tone of the whole digestive system is lost in course of time. Even in such a case, by the gradual reduction of the dose of castoroil and subsidising its action by the addition of fruit laxatives such as grapes decoction or mild aperients, the tonic action of the bowel may be restored and gradually the castor oil habit must be cut off and the bowels educated to act spontaneously. In more severe cases Milk of Magnesia, Aloes, Cascara Sagrada, or Phosphate of Soda (5–15 grains for each feed) have to be tried. The following is an effective mixture.

℞	Tr. Aloes	... ℥ v
	Sodium Sulphate	... gr. x
	Tr. Belladonna	... ℥ i
	Ext. Cascara Sargada liq.	... ℥ x
	Syrup Zingiberis	... ℥ xx
	Aqua Menth pip ad	... ℥ i

The dose of this mixture must be adjusted in each particular case, just sufficient to have a liquid motion. If necessary it may be repeated in the evening. It should be given for a number of weeks and it should be gradually discontinued when it is thought that the bowels would act spontaneously. In more obstinate cases Syrup Senna 15–20 minims or Tincture Podophyllin 1–2 minims will be useful. At times more stubborn cases may be met with when none of the above remedies will be of any use. The writer has seen a child of 4 years in whom even 6 drams of Mag-Sulphas could not produce a single motion and the child was passing hard, chalk-like pellets once in 8 or 10 days. In such cases, Picrorrhiza will be very effective. About 2 ounces of decoction are to be extracted from one ounce of Picrorrhiza Rhisomes (Tamil-*Katugurogam* and Telugu-*Katukarogini*) and $^1/_2$ to 4 drams of the fresh decoction may be given once a day. Grapes decoction also must be used side by side.

When the stagnation is in the lower part of the large bowel only, suppositories and oil enemas may be of some use. But this should not be used as a routine measure in chronic constipation, as this does not stimulate the bowel or increase its tone to respond spontaneously and hence it continues to remain constipated.

COMMON AILMENTS AMONG INFANTS AND CHILDREN AND THEIR TREATMENT

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BEFORE I proceed to discuss the common ailments, I must state some of the general points of importance regarding the pathology and treatment of children. Pathological processes in children tend to be pure bred, they are not hybrids produced by a blending of different morbid factors. Disease in children also runs a less complicated course than it does in the adult and tends to exhibit pathological processes in their simplest forms. As regards therapeutics, the treatment of children is eminently satisfactory. Children respond to treatment in a most surprising and gratifying manner. We can modify children's surroundings. In the case of children we fight with Nature instead of against her. They are going up the hills, hence the treatment of their diseases is encouraging.

Infants are babies below the third year of age *i.e.* upto the teething period; and children are babies above that age. Practically every type of disease is found in infants and children but I will discuss only the common ailments we come across daily in the outdoor. The ailments are surgical and medical. Surgical diseases common in infants are umbilical hernia, phimosis and enlarged tonsils.

Umbilical hernia is due to neglect, carelessness on the part of the nurse at the time of ligaturing the umbilical cord at birth. The treatment is mostly palliative *i.e.*, application of suitable pad except in the case of big hernia where operative treatment is to be resorted to.

Phimosis is caused by elongated male prepuce, and neglect by the nurse in not cleaning the area, causes accumulation of perpetual secretion underneath it causing source of irritation. The treatment consists in pushing the prepuce by force and lubricating the area with Liquid Paraffin for a couple of days. If the prepuce is fixed and does not retreat circumcision is the only alternative.

Enlarged tonsils.—Majority of infants and children have enlarged tonsils. This is always a physiological one and not pathological because tonsils help in the growth of the child and in majority of cases they are seen contracting and squeezing in due course. So don't insist on removal of the tonsils in every case except when the tonsils are septic and there remains temperature or in the case of chronic otitis media.

Medical ailments.—Majority of the medical ailments belong to the gastro-intestinal system, and the respiratory system. In addition to these, many suffer from nutritional and skin diseases.

Digestive disorders:—Most common of them are colic, vomiting, diarrhœa and constipation. All these symptoms are mostly due to

irregularity in diet, *i.e.* mother does not know when and how much milk is to be given. Mother, on screaming of her infant, thinks him to be hungry, gives milk, both her own as well as artificial, which produce overfeeding, causing colic, vomiting and diarrhœa at various stages. Infants usually scream (1) due to overfeeding, never by starvation; (2) due to ear-ache; (2) due to teething; (4) due to phimosis; and (5) due to mental deficiency.

TREATMENT OF COLIC.—(1) Remove the cause; (2) give warmth to abdomen; (3) hot water enema; (4) give carminative mixture; (5) if breast-fed regulate the feeding every three hours, followed immediately by sips of warm water; (6) for bottle-fed babies clean the bottle properly, always use two bottles with separate nipples and teats. Use them alternately. Always give warm water after every feed. Feed at regular intervals after every three hours. Carminative mixture commonly used is Soda Bicarb gr. ii, Spt. Amm. Arom. m. ii, Glycerine m. ii, Aqua Menth Pip every two hours to be repeated. If colic still remains add Chloral Hydras gr. i, and Tr. Opii. m. i in the above mixture.

Vomiting is either acute due to gastric catarrh, or chronic due to irregular unsuitable feeding.

TREATMENT OF VOMITING.—Keep the infant warm. Give plenty of warm water. Stop the feeding for some time. Give carminative mixture with Vin. Ipecæ m. ii, Pot. Bromide gr. ii, Tr. Card CO m. iv. Give small doses of Sulphaguanidine.

In cases of chronic vomiting always regularise the feedings. Give hot water after each feed. If vomiting takes place immediately after feed, reduce the size of feed, prolong the intervals. If vomiting occurs half an hour after feed and consists of curd then give carminative mixture with Sulphaguanidine powders in small doses. If vomited matter is very sour producing stomatitis give Vitamin C injections 1 c.c. with alkaline carminative mixture. If everything is vomited and there is general debility accompanied by progressive loss of weight, give Glucose iv and stop everything by mouth. Give Normal Saline subcutaneously.

Diarrhœa is very common in infants. Main causes are:—(1) Infant's stomach is small and contains less of hydrochloric acid; (2) diet is mostly milk which is usually given in excessive quantities; (3) infants are particularly liable to chill; (4) dirty surroundings, foul drains, contaminated soils, overcrowding, dirty cesspools, dung heaps etc. Diarrhœa may be acute and chronic. In the acute type the motions are more frequent, there is fever, vomiting, stools are yellow then greenish, there comes mucus, blood and lastly they are offensive and watery. Green stools are due to the growth of special micro-organisms and the presence of bile pigments. Putrid offensive stools are due to peristalsis in the upper portion of intestines being too rapid, so imperfectly digested and incompletely absorbed food

comes in the colon where putrefactive organisms grow. Lumpy cheesy stools are due to undigested milk being taken in large quantities. Irritating stools are due to development of fatty acids. Frothy stools are due to taking too much sugar. Slimy stools contain too much mucus and if it contains blood indicate involvement of large intestines.

TREATMENT OF DIARRHOEA.—Most important principles are starvation, elimination. So starve the child. Stop milk. Give plenty of boiled water, glucose water. If child is dehydrated, give subcutaneous injections of Normal Saline and Glucose 25% intravenously. If pulse is weak give Cardiamid injections. If motions are very sour give Alkaline mixture. If stools are slimy and alkaline give Acid mixture.

℞ Acid Sulph. Aromat	...	℥ ii
Liq. Hydrarg Perchlor.	...	℥ ii
Tr. Card Co	...	℥ iii
Spt. Chloroform	...	℥ ii
Aqua ad	...	℥ i 4 hourly

If stools are green give Castor oil Emulsions:

℞ Ol. Ricini	...	℥ v
Mucil: Acacia	...	q. s.
Spt. Chloro	...	℥ iv
Aqua Menth pip ad	...	℥ i
℞ Calomel	...	gr. ½
Dover's powder	...	gr. ½ t.d.s.

If stools are watery and offensive :—

℞ Silver Nitras	...	gr. 1/6
Acid Hydronitric dil	...	℥ ii
Glycerin	...	℥ v
Aqua ad	...	℥ ii 3 hourly

If stools are very frequent and accompanied by much straining and in lienteric type of diarrhœa *i.e.* in which motions tend to occur immediately after food is introduced into the stomach, add Tr. Opii or Chlorodyne ℥ ii to above mixtures. But don't give Tr. Opii at the onset of the disease. Don't give it when the heart is sinking. Don't give it when the tongue is furred. Never wake the child up to give medicine containing Tr. Opii. In chronic type of diarrhœa give Enterovioform, Enteroquinol, Formo Cibazol, Sulphaguanidine. Some cases of diarrhœa where stools are too many a day but the child does not seem to become weak and where every precaution regarding regularity of diet, time etc. has been observed, improve spontaneously with

℞ Acid Phosphoric dil	...	℥ vii
Aqua ad	...	℥ i 4 hourly

Resistive types of diarhœa of chronic nature improve by giving injections of Celin and Peloninamide on alternate days.

Dysenteric type of diarrhœa responds well to Stovarsol, Carbarsone and intramuscular injections of Emetine. It is useless to give Emetine in small quantities at long interval.

Give $^1/_2$ gr. every day for five days only. In case of diarrhœa alternate with constipation give Cremosuxidine Emulsion.

Constipation.—Chronic constipation is commoner in breast-fed than bottle-fed infants because there is less quantity of fat in mother's milk. Constipation is due to less amount of intestinal secretion, so stools become hard and friable.

TREATMENT.—Never indulge in purgatives except when absolutely essential. Give few doses of Mist. Alba to clean the bowels followed by fluid milk of Magnesia or Petrolagar. In obstinate constipation.

℞	Soda Sulph	... gr. x
	Tr. Aloes	... ℥ iv
	Tr. Belladonna	... ℥ i
	Syrup Senna	... ℥ xx
	Aqua Menth Pip ad	... ℥ ii t.d.s.

If there is straining with hard stools give Confectio Sulphuris in half teaspoonful doses at bed time. Give plenty of liquids to drink. If liver is sluggish—

℞	Pulv. Hydrarg. c Creta	... gr. i
	Soda Bicarb	... gr. iii

at bed time and followed by the mixture as below during day :—

℞	Soda Sulph	... gr. x
	Tr. Aloes	... ℥ v
	Elixir Cascara	... ℥ xv
	Syrup Senna	... ℥ xx
	Aqua Menth Pip ad	... ℥ ii t.d.s.

If there is mucus and worms, add Pulv Rhei gr. 5 to above. Give Elixir Vitamin B complex to regularise the bowels.

Respiratory ailments.—Bronchitis is a very common disease in the infants and children because they get chill soon due to large body surface, other causes being rickets and teething. This disease should be carefully treated because once it starts and is not properly checked it will produce various complications like bronchopneumonia, tuberculosis etc.

TREATMENT.—Give plenty of hot drinks. Apply Lint Terebenth on the chest, back and neck. Apply anti-congestin poultice over the back only, never over the front of the chest at all, because that will further labour breathing. Prescribe :

℞	Soda Salicylas	... gr. v
	Sodæ Bicarb	... gr. v
	Liq. Am. Acetas	... ℥ v
	Spt. Am. Arom.	... ℥ v
	Ext. Glycerrizæ Liq.	... ℥ xx
	Syrup Tolu	... ℥ xx
	Aquæ Anisi ad	... ℥ iv, ¼ 4 hrly.

Give small doses of Sulphadiazine with Soda Bicarb alternate with the mixture. Add Tr. Ipecac if there is thick mucus and furred tongue. Don't wait for the complications to set in, check the disease in the early stage, so give Distaquine injection (Procaine Penicillin) 100,000 to 300,000 depending upon the age, every day, for a couple of days. Always give Penicillin in high concentration.

Pneumonia is a common complication of bronchitis. Both types are found *i.e.*, lobar-pneumonia and broncho-pneumonia. Broncho-pneumonia is mostly found in infants and lobar-pneumonia in children.

TREATMENT.—Give plenty of fluids. Apply Lint Terebenth over the chest and back. Apply anti-congestin to the back. Give the mixture as in bronchitis, or

℞		
	Pot Iodide	... gr. i
	Soda Salicylas	... gr. v
	Soda Bicarb	... gr. v
	Tr. Lobelia Etheris	... ℥ v
	Elixir Cascara	... ℥ xx
	Aqua ad	... ℥ iv, ¼ 4 hrly.
	Sulphadiazine	... Tab. one
	Sulphamerazine	... Tab. one, ¼ 4 hrly.

Give injections of Distaquine 100,000 to 300,000 morning and evening till the breathing improves. Keep the patient warm.

Nutritional diseases.—Most common diseases are marasmus and rickets.

Marasmus.—This is a common nutritional disease. Causes are: (1) Organic diseases *e.g.*, tuberculosis, congenital diseases of the heart, repeated attacks of broncho-pneumonia; (2) starvation, especially in case where mothers die immediately after birth of child or child is unable to suck; (3) premature infants; (4) improper and irregular feedings; and (5) dyspepsia *e.g.*, vomiting diarrhœa.

TREATMENT.—Keep the child warm. Put the child on breast milk if possible. If not then put him on buffalo's milk 1:3 or Glaxo milk every three hours followed by hot sips of water. Give Calcinol Granules Siotrat Liver Extract by mouth. Put the the child on Adexolin, Elasmin liquid, Thyroid tablets. Give occasionally grey powder.

INJECTIONS.—Give Collosol Calcium with Vitamin D (Crooke's), injections alternate with whole Liver Extract Crude (T.C.F.) on every alternate day. Give 2 c.c. of each at a time because small doses do not give good and effective results. Expose the baby to ultra-violet rays.

Rickets.—Another common nutritional disease. It is more commonly found in cities than in villages; because of overcrowding, lack of sunlight, also because the women do not nurse their children and put them on artificial diet from the very start.

TREATMENT.—Regularise the diet of child. Treat digestive disorders *i.e.*, diarrhœa, loss of appetite etc. Put the child on mother's milk if possible, otherwise on buffallo's milk 1:3 every three hours. Don't give milk at night. Give Ostelin liquid by mouth. Give injections of Ostelin Fortis, Vitamin A and Collosol Calcium with Vitamin D. Give ultra-violet rays. Let the child spend most of his time in open air and sunlight.

Skin diseases.—Various types of skin diseases are found in children and infants.

Pemphigus.—In this disease large number of bulæ appear in the body. If they appear also on soles and palms then it is syphilitic, otherwise non-syphilitic.

TREATMENT.—In syphilitic type, give N.A.B. and Acetylarson injections. In non-syphilitic, give injections of Urea Sulphazide. Locally Acriflavine 1% dressing.

Intertrigo is the fretting of the skin due to some superficial irritation *e.g.*, by excreta, so it is found in the parts covered by napkin, or irritation by decomposition of the secretions of the skin where two cutaneous surfaces meet *e.g.*, behind the ears, in axilla, groin and neck. Recently a case of imbecility came having intertrigo in the folds of the skin of both thighs.

TREATMENT.–Avoid soap. Apply Calamine dusting powder. For intertrigo behind the ear apply Silver Nitras in Spt. Etheris Nitrosi 2%. In the case of napkin intertrigo reduce the amount and frequency of feedings, stop starch foods, and treat diarrhœa. Change the napkins as often as soiled. Apply Zinc Oxide, Acid Boric and Calamine (Z.A.C.) powder, locally. If sepsis occurs in intertrigo give a few injections of Penicillin. If watery discharge appears give Urea Sulphazide injections.

Eczema is another skin disease with serious exudate trickling out of it. It is more commonly found in infants during teething periods.

TREATMENT:—Internally give Pot. Iodide, Pot. Citras, Tr. Guaiacum, or Quinine. Locally apply Z.A.C. powder dissolved in hot water in thick paste. If septic, give Distaquine injections followed by Aektisol 2 c.c. injections.

Seborrhoeic dermatitis.—Starts mainly on the scalp and as grossy yellowish flakes of scales.

TREATMENT.—Remove the scales with 2% Salicylic ointment and give Urea Sulphazide injections, and if septic give injections of Distaquine. Wash the scalp daily with warm milk followed by warm water after an interval of twenty minutes daily.

Pruritus ani and vulva is due to hæmorrhoids, oxyuris vermicularis, vaginal or anal catarrh. Treatment consists in the removal of

the cause. If pruritus is of long duration and has produced pseudo-condyloma give N.A.B. ·15 gm. weekly injections and Acetylarsan 2 cc. on alternate days.

Scabies is very common in infants and childhood. It was in an epidemic form in 1947, after the partition of the country. Not a single displaced family was free from it. It usually occurs at the angles of fingers and toes. In dirty atmosphere it takes on various forms, sometimes covering the whole body resembling psoriasis.

TREATMENT.—Clean the body with Tetmosal soap. Apply Tetmosol ointment or lotion over the body. Apply Sulphur ointment over the hands and toes. Give Urea Sulphazide 2 c.c. or Aektisol injections 2 c.c. intramuscularly on alternate days.

I have given above some of the common ailments infants and children suffer from, but I am not going to discuss various types of fevers children suffer from *e.g.*, malaria, typhoid etc., because they form separate entity.

ANKYLOSIS JAW IN CHILDREN

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ANKYLOSIS of jaw occasionally occurs in children and then results in failure of mandibular development and deformity of the face, which is bird-like with protruding nose and upper lip. The chin remains retracted and depressed. There is a great tendency for these patients to develop oral sepsis due to the lack of the movement of the jaw.

The causes of the ankylosis may be due to intra-articular or extra-articular fibrosis.

The extra-articular causes are usually peri-articular infections due to infective parotitis and cancrum oris. X-ray of the mandibular joints excludes any intra-articular lesion.

CASE NO. 2 showed the X-ray appearance of a normal joint but after excision of the condyles and opening the mouth a sequestrum was removed from the body of the mandible. Here the osteomyelitis of the mandible gave rise to the extra-articular fibrosis of the tissues and muscles surrounding the joint.

The intra-articular ankylosis is the result of suppurative arthritis of the mandibular joint, which is affected either primarily or secondary to suppurative parotitis as in Case No. 1 or pyæmia. Sometimes it may be due to fracture of the condyle or dislocation of the joint or displacement of a torn miniscus.

The patient with the ankylosis of jaw due to the causes above described is in a good general state of health and without any affection of the general nutrition. There is adequate intake of

fluid diet in the form of milk soup, fruit juices, and the use of a straw pipe facilitates the taking of fluid nourishment.

It is only the hygiene of the mouth and teeth which cannot be looked after and so the teeth get septic and decay. The condition of chronic trismus should be distinguished from the acute condition of a risus sardonicus due to tetanus and that due to acute suppurative arthritis and parotitis. A historical condition of trismus has been described in young girls and a reflex variety due to impacted 3rd molar, or infected molar teeth. An irritative ulcer on the gums or floor of the mouth may also give rise to reflex trismus.

TREATMENT.—Where the cause of ankylosis is extra-articular, the jaw should be opened under anæsthesia. If this treatment fails then Esmarch's operation of removing a wedge of triangular bone from the mandible in front of its angle may be done. If the X-ray reveals bony ankylosis of the mandibular joint then excision of the condyle of the mandible should be done on both sides. This gives very good results and the jaw starts opening immediately. In both of my cases, where the operation was done the patients started eating the next day. In Case No. 1 the synostosis of the condyle with the base of skull was so dense that the chisel had to cut through the base of the skull to separate a piece of dense thick bone from the condyle on the left side of the mandible. The operation may only be required on the affected side but sometimes it has to be done on the opposite side as well. I perform the operation on one side and if the mouth does not open properly the excision of the opposite condyle is also done. The operation of the excision of the condyle is preferred to Esmarch's operation. The operation is done by a horizontal incision 1" long along the lower border of the zygoma at the level of the tragus. The temporal branches of the facial nerve are drawn aside and the parotid gland downwards. The joint is exposed by a vertical incision after dividing the masseter muscle from the zygoma.

If the condyle is dislocated it is either pushed back in position or cut away.

In the presence of bony ankylosis, the condyle may totally disappear, or there is no sign of a joint as in Case No. 1.

Here enough bone is excised as to create a gap for the formation of a false joint. There is no necessity of an arthroplasty as the operation of simple excision helps to establish mobility of the lower jaw.

An after treatment of active and passive movements of the lower jaw is carried out and the patient is encouraged to eat solid food.

CASE NO. I.—A Hindu female patient, named J.D., aged 10 years, was admitted with the complaint of inability to open the mouth for the last one year. She was only able to take milk as her nourishment.

Previous history:—She gave a history of boils in the post-auricular region, where there were scars present.

On examination there was a deformity of her jaw, the mandible not having developed fully. She had a retracted chin, the lower lip receding behind the upper.

The left side of the temporomandibular joint appeared to be a soid mass beneath the skin without any outward deformity or scarring. There were scars present in the post-auricular region.

X-ray of the left temporomandibular joint revealed synostosis of the articulating surfaces of the joint and was not clearly seen. The right jaw was normal.

Operation notes:—Under general anæsthesia a transverse incision was made beneath the lower border of the zygoma in front of the tragus. The fibres of the masseter muscle were cut and also the capsule of the joint and a broad condyle of the mandible about 1" wide was seen firmly united to the articular surface beneath the base of the skull. By chisel and hammer, pieces of bone were removed till a space in which a forefinger could be passed was created between the remaining portion of the condyle and the base of the skull.

The mouth could be opened slightly. The condyle of the right side was also removed through the latter was normal. Now the mouth could be opened sufficiently wide. The wounds, which exposed the joints, were stitched and healed by primary union.

Discussion.—The patient had a very firm bony union of the left temporomandibular joint with broadening of the condyle. The cause could only have been a suppurative arthritis of the joint in childhood probably secondary to left parotid abscess, which burst posteriorly below and behind the left ear.

CASE NO. II.—Patient, named D. J., Hindu, male, aged 9 years, was admitted with the complaint of inability to open his mouth for the last 9 months. The child suffered from typhoid fever about ten months ago and during its course developed swelling over the face first on the left side and then on the right. An abscess developed which burst on the left side of the face. Since then he is unable to open his mouth.

Physical examination:—The patient is totally unable to open his mouth. He can take only fluid nourishment. There are a few scars present over the left temporomandibular joint, which is slightly more prominent and painless.

X-ray report:—X-ray report did not reveal any synostosis of the articulating bones of the joint.

Operation notes:—Under general anæsthesia a transverse incision was made over the left temporomandibular joint in front of the left tragus. The fibres of the masseter muscle arising from the

zygomatic bone were cut and so the capsule in the line of the incision. The mandibular joint was opened and the condyle of the mandible was removed. The jaw could not be opened. The same operation was done on the right side when the mouth could be opened easily. On opening the mouth there was a foul smell and a sequestrum was found on the alveolar edge of the left side of mandible near the molar teeth which was removed. The wounds over the joints were stitched. The patient started opening the mouth from the day of operation and he was encouraged to eat solid food. Hydrogen Peroxide gargles were given twice a day and Potassium Permanganate gargles several times and between. The stitches were removed on the 9th day and he was discharged cured.

Discussion.—The patient developed ankylosis of the jaw probably secondary to bilateral parotid abscesses, which burst outside as well as inside the joint, thus giving rise to suppurative arthritis. There was osteomyelitis of the mandible also with formation of sequestrum, which would not have been evident unless the mouth could have been opened by excision of the condyles. The whole pathological process was secondary to complication of typhoid fever.

Summary.—1. Two cases of complete bony ankylosis in children have been reported.

2. The operative treatment of the condition is described; which should be done as early as possible by excision of the condyles.

This operation is preferred to Esmarch's operation.

BOOK REVIEWS

"Medicines for Students"—By A. F. Golwalla. M.D., F.C.P.S. with a Foreword by Dr. Roustom Jal Vakil, M.D. (Lond.), M.R.C.P. (Lond.), D.T.M. & H. (Lond.), F.R.F.P.S.G., F.C.P.S., F.A.SC., Honorary Physician, K.E.M. Hospital, Bombay. Total number of pages 684. Copies of the book can be had from M/s. Current Technical Literature Co. Ltd., 'Jehangir Buildings,' 133, Esplanade Road, P.O. Box 1374, Bombay 1. Price Rs. 16/-

This book has been written for the medical students. The plan of presentation is that followed by many clinical teachers in the wards. It is divided into 12 chapters. Each chapter deals with a particular system and diseases occurring in that system. Every chapter has been dealt with in a clear and concise manner, the subjects having been classified in a tabular form wherever possible. There are special chapters on Tropical Diseases and Diseases of Children. Subjects such as Vitamins, Allergic Manifestations, Pathology of the Blood, Examination of the Fundus, Anti-biotics, Virus Diseases are dealt with in separate chapters. This book is a very good synopsis of medicine, and will be specially useful to the students of medicine and the busy general practitioner who will find in its pages essential matter for daily use.

The printing and get-up are good. We congratulate the author and recommend the book to those who seek to teach clinical medicine and to the student who is appearing for his examination.

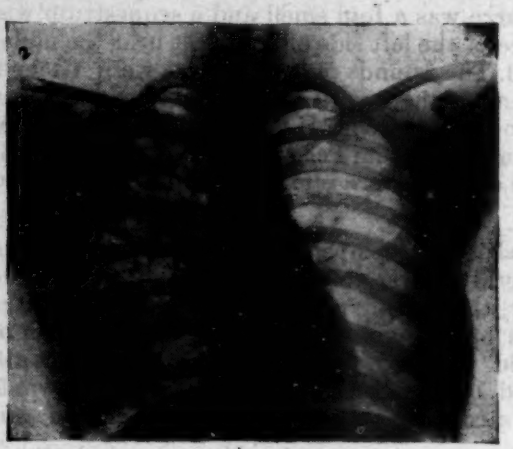
U.K.B.

Land Mark—By Dr. M. C. BHOWAL, L.M.P., (Binapani Book Dept. 22, Gonendra Mitra Lane, Calcutta), 38 pages. [Price Re. 1

This book gives surface markings of important structures in the human body. The first part deals with surface markings of bony points, sinuses, trephine points in the head and neck. The surface markings of important structures such as the lungs, heart, arteries, liver, stomach, pancreas, important blood vessels are then given. The surface markings of grooves, fissures, foramen and canals are then given. The book will serve a very useful purpose.

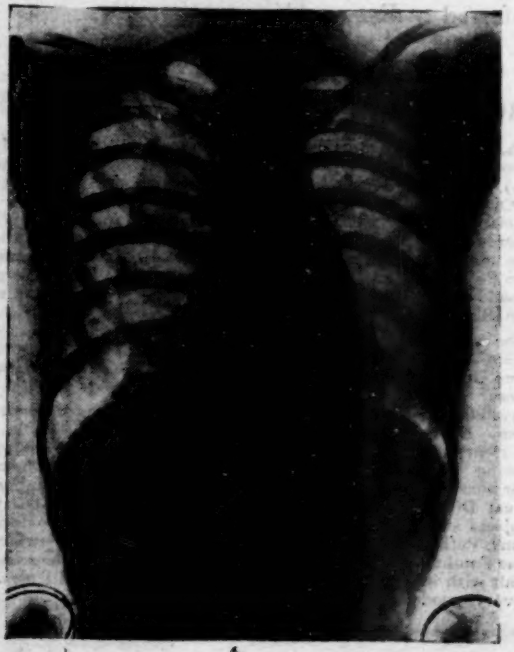
Corrigendum

In the article "Chloromycetin As An Anti-biotic" published in the June 1951 issue of the ANTISEPTIC, in page 465, the X-ray photographs were misplaced and wrong write-ups given by mistake. They should be as under:



X-ray before treatment.

Extensive exudative lesions in the right lung. Left lung comparatively free. The cardiac shadows normal, diaphragm normal. Suggestive of tubercular infiltration in the right lung.



Skiagram showing right lung partially collapsed. Left lung normal.

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